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TULAREMIA IN NEW ENGLAND

A Review of Eighteen Cases, with the Report of Two Additional Cases

FRANCIS D. MOORE, M.D.,* CARL S. SAWYER, M.D.,† AND S. GILBERT BLOUNT, JR., M.D.‡

BOSTON, MASSACHUSETTS, AND PROVIDENCE, RHODE ISLAND

THE term "tularemia" has been applied to a wide variety of illnesses due to human infection with *Pasteurella tularensis*. The most frequently occurring clinical picture is that of an open ulceration with regional lymphadenopathy and evidence of severe systemic infection that appears to be out of proportion to the local lesion. This ulceroglandular form of the disease may be accompanied by pneumonic involvement, rash or enlargement of the spleen. The infection may also produce a syndrome in which the systemic factors entirely outweigh the local lesion, in which event the typhoidal form of the disease is found. The organism responsible for this infection is apparently endemic in a fairly widespread animal reservoir and is transmitted from animals to human beings either by an insect bite, by direct handling or by ingestion of infected animal material.

The ulceroglandular form of tularemia so closely resembles various trivial nonspecific peripheral infections and infected lacerations that it is often overlooked. Furthermore, the local lesion may be so minor as to be completely overshadowed by the systemic manifestations, leading to a mistaken diagnosis of blood-stream infection with gram-negative bacilli such as the typhoid bacillus or a febrile illness such as brucellosis, rheumatic fever or subacute bacterial endocarditis. It is therefore necessary to stress the importance of this disease in differential diagnosis and the need of an exhaustive search for a possible local lesion in the presence of fever of unknown origin. Because of these facts, as well as the appearance of 2 recent cases in New England, it has seemed worth while to review the history of tularemia in New England, bringing the literature on all the available cases up to date.

New England appears to have been the last region of the United States to remain free of infection with *Past. tularensis*. By 1932, tularemia had appeared in all the states of the Union except Maine, Vermont and Connecticut[1]; New England could claim the fewest cases of any sizable region in the country and contained the only three case-free states. Since that time, cases have appeared in New England at the rate of approximately 2 a year, and at the present time it is possible to bring together a total of 20 cases. Vermont remains as the only state apparently free of the disease.

Whether this phenomenon is due to an increasing incidence of tularemia in the region or merely to increasing recognition and bacteriologic confirmation is an academic question that cannot be answered on the basis of the material at hand. Although the disease was first described in animals, in California, by McCoy and Chapin in 1911,[2, 3] there is no way of knowing how widespread the animal reservoir was at that time. In 1919 Francis[4] described the human disease as occurring in Utah, approximately five hundred miles from Tulare County in central California, where McCoy and Chapin had discovered the micro-organism seven years before. This geographical trend, however, is difficult to evaluate in the light of the fact that by 1938 over 1000 cases had been reported[5] from Russia, apparently contracted from the skinning of water rats, and many other cases had been found in Japan, Norway, Canada, Sweden and Austria. By these data one is forced to the conclusion that although American in origin, scientifically speaking, the disease is of world-wide distribution.

In the light of these facts it is interesting that one region in America should apparently stay free of tularemia until 1929. Furthermore, the grouping of the New England cases indicates that factors of importation, either of rabbits or of infected human beings, seem still to be operative in the spread of the disease. This corroborates the impression that tularemia is still a relatively new disease in New England, not only to human beings but also to animals.

These factors of importation concern firstly 3 patients in the Boston area, one who contracted the

*Assistant in surgery, Massachusetts General Hospital, and instructor in surgery, Harvard Medical School, Boston.

†Graduate assistant in dermatology, Massachusetts General Hospital, Boston.

‡Intern, Medical Service, Rhode Island Hospital, Providence, Rhode Island.

disease in Colorado and then came to Boston, a second who contracted it from tick bites while in Georgia and a third who contracted it from a rabbit shot in Illinois and shipped here. Furthermore, in 1936, cottontail rabbits were imported from the West and released near Falmouth, Massachusetts, and within two years 3 cases had been reported from that district. A fourth case occurred in the region of Falmouth in 1943. This makes a total of 7 cases in New England — nearly a third of the entire series — directly traceable to importation of infected animals or contracted elsewhere. It is impossible to say to what extent these importations have influenced the further spread of the disease in New England, but the fact that 4 more cases have occurred within seventy miles of the Falmouth area is suggestive.

Chronological Review of New England Cases

The first case to appear in New England was reported from Massachusetts by Withington.[6] The patient contracted the ulceroglandular type of disease from tick bites while visiting on an island off the coast of Georgia in 1929. He had a septic abrasion on the scrotum, with an enlarged inguinal node that subsequently suppurated, and x-ray evidence of a slight pneumonitis on the left side. Widal and Wassermann reactions were negative, but the serum agglutinated *Past. tularensis* in a dilution of 1:1280 and *Brucella abortus* in a dilution of 1:160 (partial agglutination in a dilution of 1:320). Randall[7] in 1929 reported a case in Rhode Island in which a man contracted tularemia after handling three dead rabbits. A cat that ate one of the rabbits also contracted the disease and died. Agglutination of the patient's serum against *Past. tularensis* was positive. The second case to appear in Massachusetts[8] occurred in Boston in 1929. The patient evidently caught the disease from a cold-storage rabbit that had been frozen for thirty days and imported from Illinois. The patient had ulceroglandular tularemia with an urticarial eruption, and serum agglutination against *Past. tularensis* was positive in a dilution of 1:640.

Badger[9] and Eckstein[10] both referred to 2 cases, one occurring in New Hampshire in 1931 after the handling of rabbits and the other in Maine in 1933 after the skinning of a fox. Further details on these cases have not been found elsewhere in the literature, but the second one has been confirmed by the Maine Department of Health and Welfare.[11] The disease in this case was of the ulceroglandular form and was fatal. It was proved by autopsy, culture and agglutination.

In 1934,[12] a man contracted the ulceroglandular form of tularemia from the ingestion of a rabbit that he had killed on a ranch in Colorado. He returned to Boston after his symptoms had appeared and was hospitalized. Serum agglutination was positive in dilutions up to 1:1280, and a skin test with the Foshay tularemia suspension was positive.

In 1935 and 1936, 3 cases were reported to the New Hampshire Department of Health.[13] All these were clinically stated to be tularemia and accepted as such by the state authorities, but the details and bacteriologic report are not available.

In 1937,[9] a ten-year-old girl developed the typhoidal type of tularemia in Falmouth, without skin lesions or adenopathy, presumably from the saliva of her dog, which became ill three days later. Specific agglutination was positive in dilutions of 1:1280. The dog serum was positive in dilutions up to 1:40. This case appeared three months after Western cottontail rabbits had been released in the neighborhood of Falmouth. In the same district, one year later, another child developed ulceroglandular tularemia after being bitten by a tick.[12] The bite was in the scalp and was accompanied by enlarged postaural nodes. Serum agglutination tests were not done. A Roxbury resident visiting on Cape Cod in July, 1939, was bitten on the thigh by a tick, subsequently developing ulceroglandular tularemia with pneumonic symptoms.[14] This patient, unlike the others, had severe systemic disease and serum agglutination tests were positive in dilutions up to 1:1280.

Gibbons, Lamoureux and Arkless[15] described a case in Connecticut that occurred in December, 1940. After killing and dressing three rabbits the patient developed tularemia with a finger ulcer, chills, fever and adenopathy, which subsided in three days. A relapse eleven days later with severe systemic disease proved fatal. Agglutination tests were positive in dilutions of 1:640.

In 1941, a man in Lawrence, Massachusetts, complained of an abrasion on his finger that subsequently became necrotic. The diagnosis of tularemia was established by history of exposure to rabbits that he had shot and dressed, as well as by the findings of adenopathy, chills, fever and a specific agglutination test in serum diluted to 1:640.[12]

A second case was reported from Rhode Island by Eckstein[10] in 1941. The patient had the ulceroglandular form of the disease, contracted after dressing a rabbit that he had shot. The primary lesion appeared in a puncture wound of the hand sustained two days before. Specific agglutination was positive in serum dilutions up to 1:2560. This patient received sulfanilamide therapy.

Two other cases were reported from Connecticut in 1941. The first of these[16] occurred in Columbia, only ten miles from Colchester, where the previous Connecticut case had occurred.[15] This was a fatal case of ulceroglandular tularemia, with pneumonic manifestations, contracted from a cat bite. The second was a case of ulceroglandular tularemia from handling rabbits,[17] it occurred in Stamford. Agglutination tests were positive in a titer of 1:80.

The most recent New England case to appear prior to the 2 reported below was a fourth case from the Falmouth district,[12] following a tick bite. The patient developed right-sided pneumonia without evidence of a primary lesion or adenopathy. Specific serum agglutination tests were positive.

Report of Cases

Case 1. The patient (M. G. H. Unit 405941) was a 52-year-old inhabitant of Waltham. He was admitted to the Emergency Ward on June 8, 1943, and stated that 3 weeks before, while working in his garden, he had been bitten over the upper sternum by a small insect. He had not noticed the bite until he went into the house and looked in a mirror. He did not identify the insect, but when he removed and crushed it, it appeared to be distended with blood and to have a hard shell.

For 3 days nothing further was noted about the incident. At the end of that time the patient began to notice occasional chilly sensations, fever and considerable muscular weakness. Following a week of such symptoms, not severe enough to incapacitate him, he noticed soreness in the left axilla and a small papule at the site of the bite, which broke down and formed an ulcer that gradually increased in size. He poulticed the lesion and called in his physician, who gave him sulfadiazine totaling 3 gm. a day for 10 days. Since the fever and malaise persisted, as well as the ulcer and the sore node in the left axilla, he was referred to the hospital.

On entry to the Emergency Ward the patient had a temperature by mouth of 101.8°F. and a white-cell count of 10,400. The urine was negative. The ulcer over the manubrium was 1.5 cm. in diameter, with a 3-cm. zone of erythema surrounding it. It was not tender and there was no undermining or penetration, but the edge was indurated. Little exudate was present, it was not granulating, and there was no evidence of undrained pus. A firm, tender, freely movable lymph node the size of a walnut was present in the left axilla.

The patient had had no contacts with rabbits, ground squirrels or other rodents and the only small animals he had seen around his garden were chipmunks.* He did not remember seeing any that appeared ill, and no dead ones had been found to his knowledge.

Tularemia was suggested as a tentative diagnosis, and the patient was admitted to the hospital. Primary syphilis, symbiotic staphylococcus-streptococcus infection, anthrax, sporotrichosis and other mycotic infections were also considered. A blood culture yielded no growth, and serum agglutination tests against *Past. tularensis* done on admission were negative in all dilutions. Cultures from the ulcer, taken on the usual aerobic and anaerobic media, showed a coagulase-negative hemolytic *Staphylococcus albus*, diphtheroids and aerobic spore-formers. In addition, some yeasts were found that were cultured and studied for virulence by mouse injection, with negative results. Dark-field examination of the material from the ulcer for *Treponema pallidum* was negative. The blood serum gave a positive Hinton and a negative Wassermann reaction on two occasions. A Hinton test the day before discharge was negative. Agglutination tests for typhoid and paratyphoid fever were negative. Stool and urine cultures were negative.

The clinical course was one of continued swinging fever, as high as 103.6°F., for the first 10 days in the hospital. The patient had few symptoms other than malaise. The white-cell count fell to 6000 by June 22 and he consistently showed a high monocyte percentage. He developed a node in the right axilla on June 11, and for 3 days thereafter he had generalized lymphadenopathy, the nodes being almond-sized, nontender and freely movable. There was no rash.

On June 21, to obtain material for histologic study to clarify the diagnosis, the ulcer was excised under Pentothal sodium anesthesia and the wound was closed with silk. The operative site healed cleanly, and the temperature immediately became normal and remained so until discharged 7 days later. The palpable nodes disappeared in 4 days.

*It is of interest in this connection that Zinsser[18] states that chipmunks are among the more susceptible group of animals to infection with *Past. tularensis*. Welch and Jakmauh[19] point out that the chipmunk is also one of the common hosts in this region for the wood tick *Dermacentor variabilis*.

The pathological report from the excised lesion was "acute and chronic inflammation," with no changes characteristic of either syphilis or other specific granulomas. Blood serum taken for heterophile agglutination on the same day as the operation was negative.

A repeated agglutination test for *Past. tularenis* showed the following results: serum diluted 1:20, +++; 1:40, +++; 1:80, ++; 1:160, ++; 1:320, ++. This positive agglutination, in the presence of a clinical picture compatible with the disease, established the diagnosis of the ulceroglandular form of tularemia. The fact that the earlier agglutination test was negative was attributed to its being taken too early, although the patient was in his 3rd week of the disease. He was discharged home on the 7th postoperative day. He was seen 6 months later and was in excellent health. Blood Hinton and Wassermann reactions were negative on two occasions at about that time. Serum agglutination tests of *Past. tularensis* were still positive in dilutions up to 1:320.

Case 2. The patient (Rhode Island Hospital 375228), a 36-year-old loom fixer, entered the Rhode Island Hospital on November 16, 1943. For 4 days prior to the onset of his illness on October 28, he had spent part of the day hunting rabbits; he had killed ten, skinning and dressing them himself. At the time, he had a hangnail on the left thumb, and 1 day after the onset of the systemic illness the thumbnail became infected and the inflammation persisted despite treatment.

On the day of onset of his illness the patient was awakened at about 2 a.m. by chills and chilly sensations that continued the rest of the night. Alternating with the chills, he felt warm, perspired copiously and had a severe headache. The next day he noticed the infection near the left thumbnail, and within a short period of time observed sore lumps in the left axilla "as big as eggs." He called in his physician, who noted that he had a temperature of 104.5°F. For the 11 days until entry to the hospital, this fever continued rising, to 102 or 103°F. daily. Eight days prior to entry the patient developed a cough productive of moderate amounts of yellowish-white sputum without blood. This febrile illness, with malaise, headache, cough and the infected thumb and axillary nodes, continued up to entry.

On admission the patient had a temperature of 102°F. but did not appear acutely ill. Inspiratory rhonchi and wheezes were heard over the chest, with occasional rales at the left base. There was an ulcer at the angle of the nailbed of the left thumb that oozed serum, had black ragged edges, was tender and was surrounded by a tender area larger than the ulcer itself. There was a left epitrochlear node, and a plum-sized axillary node, both of which were nontender. The white-cell count was 14,700, with 43 per cent lymphocytes. A diagnosis of ulceroglandular tularemia was made, and the possibility of pneumonic involvement was raised. An x-ray film of the chest showed patchy density in both lobes compatible with bronchopneumonia. Agglutination tests done at the Rhode Island State Department of Health were positive against *Past. tularensis* in dilutions up to 1:80. Agglutination against brucella was negative.

On the 4th hospital day, with the patient continuing to run a temperature of 102 to 103°F. daily, sulfamerazine was given, beginning with a dose of 5 gm., followed by 1 gm. every 8 hours. After 3 days of treatment there was some improvement, but not until the 6th day did the temperature drop to normal. Coincident with this fall the pulmonary lesion cleared, and serum taken at that time was positive in titers up to 1:1280 and 1:2560. The thumb lesion was still unhealed and the axillary node was still enlarged and tender, with overlying redness in the skin. The patient was discharged 4 days later.

One week after discharge, the axillary node broke down and was incised and drained, and after 1 month it was slowly healing.† On December 21, 3 weeks after discharge and 2 months after onset, the agglutination tests against *B. tularensis* were positive in dilutions up to 1:2560.

During the hospital stay, cultures from the ulcer were injected into guinea pigs, which died and showed gross lesions characteristic of *Past. tularensis* infection. Bits of liver and spleen were cultured on cystine agar in an effort to isolate the organism, but the cultures were lost and the organisms were not finally identified.

†We are indebted to Dr. Wallace Lisbon, of Providence, Rhode Island, for information concerning the course of the patient after discharge.

Discussion

Francis[20] has done much of the work on the etiology, transmission and animal vectors of tularemia, and his work should be consulted for these matters. A recent article by Foshay[21] covers the clinical aspects, methods of early diagnosis and results of serum treatment. No attempt to review the literature will be made here.

Transmission from animals to man by insect bite is not uncommon.[1] The insect in Case 1 was most likely the tick *Dermacentor variabilis*, which has previously been reported as the vector of Rocky Mountain spotted fever in New England.[22] The tick *Dermacentor andersoni*, which is rarer in the eastern United States, may have been involved. The biting season of these ticks is from March to July. The other insect that carries the organism of tularemia is the horsefly, *Chrysops discalis*, which is active from June to September.

Many different animals have been found to be infected with tularemia, and the infection may be passed directly by handling or ingestion of the flesh, as well as by the parasites that are found on the infected hosts. Of the 20 New England cases, 9 involved direct handling of rabbits and 5 were traceable to tick bites, with the animal host unknown. One was the result of a cat bite, 1 of skinning a fox and 1 of handling an infected dog, and 3 were of unknown modes of transmission.

Of the 20 New England cases, 3 were fatal. One of the fatal cases was a pneumonic relapse from the ulceroglandular form[15]; the second showed a form of tularemia involving the lungs but apparently concomitant with an ulceroglandular lesion and, at autopsy, lesions throughout the body[16]; in the third the disease was of the ulceroglandular variety.[11]

Table 1. *Summary of 20 New England Cases of Tularemia.*

Year	Locale	Animal Source or Insect Vector	Type of Lesion	Bacteriologic Confirmation	Outcome	Reference
1929	Boston (acquired in Georgia)	Tick	Ulceroglandular	Agglutination (1:1280)	Recovery	Withington[6]
1929	Rhode Island	Rabbit	Ulceroglandular	Agglutination	Recovery	Randall[7]
1929	Boston (rabbit from Illinois)	Rabbit	Ulceroglandular	Agglutination (1:640)	Recovery	Watts[8]
1931	Claremont, New Hampshire	Rabbit	Unknown	None	Recovery	Badger[9] Eckstein[10]
1933	Kokadjo Lake, Maine	Fox	Ulceroglandular	Culture, agglutination and autopsy	Death	Maine Department of Health and Welfare[11]
1935	Boston (acquired in Colorado)	Rabbit	Ulceroglandular	Agglutination (1:1280) and Foshay skin test	Recovery	Massachusetts Department of Public Health[12]
1935	New Hampshire	?	Unknown	None	Recovery	New Hampshire State Board of Health[13]
1936	New Hampshire	?	Unknown	None	Recovery	New Hampshire State Board of Health[13]
1936	New Hampshire	?	Unknown	None	Recovery	New Hampshire State Board of Health[13]
1937	Falmouth, Massachusetts	(?Rabbit) to Dog to Human	Typhoidal	Agglutination (1:1280)	Recovery	Badger[9]
1938	Falmouth, Massachusetts	Tick	Ulceroglandular	None	Recovery	Massachusetts Department of Public Health[12]
1939	Falmouth, Massachusetts	Tick	Ulceroglandular and pneumonic	Agglutination (1:1280)	Recovery	Moss and Evans[14]
1940	Colchester, Connecticut	Rabbit	Ulceroglandular and pneumonic	Agglutination (1:640)	Death	Gibbons et al.[15]
1941	Lawrence, Massachusetts	Rabbit	Ulceroglandular	Agglutination (1:640)	Recovery	Massachusetts Department of Public Health[12]
1941	Rhode Island	Rabbit	Ulceroglandular	Culture and agglutination (1:2560)	Recovery	Eckstein[10]
1941	Columbia, Connecticut	Cat	Ulceroglandular and pneumonic	Culture and agglutination	Death	Jungherr[16]
1941	Stamford, Connecticut	Rabbit	Ulceroglandular	Agglutination (1:80)	Recovery	Connecticut Department of Health[17]
1943	Falmouth, Massachusetts	Tick	Pneumonic	Agglutination	Recovery	Massachusetts Department of Public Health[12]
1943	Waltham, Massachusetts	Tick(?)	Ulceroglandular	Agglutination (1:320)	Recovery	
1943	Providence	Rabbit	Ulceroglandular and pneumonic	Agglutination (1:2560)	Recovery	

A tabular compilation of the pertinent data on the 20 cases is presented in Table 1.

Early diagnosis may sometimes be established by injecting material from the ulcer into a guinea pig, and then transferring bits of the necrotic foci in the liver and spleen to a cystine-containing medium.[1] The patient's blood culture may also be positive early in the disease, but again the organisms can usually be recovered only by animal passage. Francis[20] has pointed out that direct smear and culture are unavailing. Agglutination tests usually become positive from the third to the sixth week of the disease.

Foshay[21] has described intradermal tests with suspensions of killed organisms in phenolized saline solution, which are read in forty-eight hours and

are positive during the first week of the disease. He has also devised an intradermal test using antiserum, which is read in fifteen minutes and in one case has been reported positive as early as eight hours after the initial chill, but results have not been so successful as those with the vaccine. Foshay has also developed an antiserum from goats and has reported the results of treatment in 600 cases.

In Case 1, the patient showed a false-positive Hinton test on two occasions. Six months after recovery, the serologic reaction was negative. Such a finding has not previously been reported, and its significance must await reports of other cases. Brown and Nagle[23] give the results of eighty-seven Kahn tests on 64 patients with tularemia; only one "doubtful" reaction was obtained.

The response of the Massachusetts patient (Case 1) to primary excision of the ulcer was striking, and if the results of this method are corroborated by other observers it may constitute a useful way of shortening the period of disability from the disease. He did not respond to sulfadiazine. An appraisal of the response of the Rhode Island patient (Case 2) to a sulfonamide is difficult, since subsidence of temperature required six days after starting the drug.

Summary

Eighteen cases of tularemia occurring in New England are reviewed. Of these, 8 have not previously been available in the literature. To these are added the detailed data on 2 New England cases, 1 occurring in Massachusetts and 1 in Rhode Island during the summer and fall of 1943.

The mortality for this group of 20 cases was 15 per cent.

Vermont remains as the only state in the Union free of tularemia.

A false-positive Hinton test was a feature of the Massachusetts case. Clinical subsidence following excision of the ulcer was also an unusual feature of this case.

The Rhode Island case was one of ulceroglandular tularemia contracted from handling rabbits. In addition to local and lymph-node lesions, the patient developed definite evidence of pneumonic involvement.

We are indebted to the following state officials for their willing aid in gathering together these data on tularemia throughout New England:

Dr. Mary M. Atchison, acting state health officer, New Hampshire State Board of Health.
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Dr. Roscoe L. Mitchell, director, Maine Department of Health and Welfare;
Dr. Stanley H. Osborn, commissioner, Connecticut State Department of Health; and
Dr. William P. Shields, epidemiologist, Division of Communicable Diseases, Rhode Island Department of Health.

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VITAMIN B DEFICIENCY IN PRIVATE PRACTICE*

DUDLEY MERRILL, M.D.†

CAMBRIDGE, MASSACHUSETTS

PURE, uncomplicated diseases caused by deficiency of vitamin B—pellagra, beriberi, ariboflavinosis, polyneuritis and the Wernicke syndrome—are a great rarity in New England. This is probably true in most geographical areas where famine is unknown and the dietary habits of the majority of the people are not rendered grossly inadequate by poverty or by the lack of coarsely ground cereals, green vegetables, dairy products and meat. Occurring as complications of many different types of disease, however, these diseases are being recognized with increasing frequency and are actually of far greater importance clinically, outside the endemic areas of pellagra and beriberi, than are their uncomplicated forms.

Cowgill[1] demonstrated that the tendency to develop symptoms of vitamin B deficiency varies with the metabolic rate of the body and inversely with the vitamin B intake, and that the body weight and metabolism are consequently the most important factors determining the vitamin requirement. From these fundamental laws he was able to predict or explain the clinical occurrence of vitamin B deficiencies in association with many widely different types of disease. Shattuck,[2] Wechsler,[3] and Minot, Strauss and Cobb[4] had already reported beriberi complicating polyneuritis in patients suffering from profound nutritional disturbance, either alone or as a complication of many types of chronic illness. Strauss and McDonald[5] had also pointed out that the polyneuritis of pregnancy, previously attributed to a hypothetical toxin, is in all probability merely another example of polyneuritis due to a nutritional deficiency. In a later paper, Strauss[6] elaborated on the importance of disturbance of the gastrointestinal tract as a conditioning factor in the production of deficiency states. Weiss and Wilkins,[7,8] reported beriberi heart disease as not infrequent in patients suffering from nutritional deficiency.

From the work of all the above authors it has become apparent that clinical manifestations of vitamin B deficiency should be searched for in any situation in which there is an increased metabolism, with or without an obvious deficiency of vitamin B in the diet or a disturbance of the function of the gastrointestinal tract. This may arise because of increased muscular activity, increased caloric intake, fever, hyperthyroidism, long-continued vomiting, diarrhea or dietary restriction.

It is unfortunate that in this group of cases there is great danger of overlooking the earliest symptoms and signs of impending deficiency disease because of the frequently far greater importance of the actual precipitating factor—pneumonia, malaria, cirrhosis of the liver, pregnancy and so forth.

Many authors have stated that the earliest manifestations of vitamin B deficiency are easily reversible if proper therapy is instituted, whereas later the process may progress to a stage of slow reversibility or nonreversibility in spite of substitution therapy.

I[9] have already reported 3 cases of severe dysphagia associated with profound nutritional deficiency, 1 of them mistakenly diagnosed as carcinoma of the esophagus, all of which responded to substitution therapy. In retrospect the response to therapy can perhaps be explained by the short duration of symptoms.

Goldsmith[10] searched for evidences of riboflavin and niacin deficiency in a series of 200 consecutive patients admitted to the medical services in the Charity Hospital in New Orleans; these consisting of 50 white women and 50 Negresses, and 50 white men and 50 Negroes. She found 79 cases of definite and 54 of probable deficiency disease in this unselected series. It may be considered by some that her criteria for diagnosing these deficiency syndromes were slightly overgenerous, but there can be no doubt that these diseases were of far greater prevalence in this particular group of charity patients in a Southern city than even most nutrition experts would have expected.

Because of the frequency with which cases of vitamin B deficiency of moderate or severe nature have appeared in private practice in the metropolitan area of Boston, it has seemed worth while to report some of them. These diseases are not limited to any special group,—to the natives of Labrador or the Orient, sharecroppers, chronic alcoholic addicts or the inmates of a city hospital. Furthermore, they are common complications of many serious illnesses, and their recognition is not purely of academic interest. The therapeutic implications are obvious.

CASE REPORTS

CASE 1. C. J. M., a 63-year-old widow, was first examined because of asthma, cough and vomiting. Three days previously she had had severe chilly sensations, cough and a pain in the chest. Asthma, which she had had off and on for 25 years, returned. For 3 days she had vomited practically everything she had eaten. A year and a half before this episode she had been diagnosed as having malnutrition and diverticulosis, but in spite of much dietary advice she had failed to put on any weight.

Physical examination revealed a temperature of 103°F., a pulse of 120 and a blood pressure of 130/80—18 months previously it had been 200/110. The lungs were full of asthmatic rales, but there was a patch of pure bronchial breathing over a small area of the right upper lobe anteriorly.

*From the Thorndike Memorial Laboratory, Second and Fourth Medical Services (Harvard), Boston City Hospital, and the Department of Medicine, Harvard Medical School.

†Instructor in medicine, Harvard Medical School; junior visiting physician, Boston City Hospital; associate physician, Cambridge Hospital.

The patient was hospitalized at once. The sputum contained Type 2 pneumococci, and x-ray examination of the chest showed consolidation in the right midlung. The blood nonprotein nitrogen level was 67 mg. per 100 cc. but fell to 47 mg. 2 days later. Sulfathiazole was given for 3 days, and the temperature fell to normal in 24 hours. Five days after admission she developed urinary retention, with a blood nonprotein nitrogen of 75 mg., and exhibited considerable mental confusion. For the next 3 days the patient had to be repeatedly catheterized and fluids were given in large quantities, but the nonprotein nitrogen level rose to 86 mg. and she developed sacral and pulmonary edema, with frothy sputum and a beefy-red tongue. She was given 100 mg. of nicotinic acid every 3 hours and 2 cc. of mercupurin intravenously, and fluids were limited to 2000 cc. every 24 hours. An inlying catheter was placed in the bladder.

Twelve days after admission the nonprotein nitrogen level descended to 25 mg. and the temperature to below 100°F. The patient was well oriented, the edema had disappeared, and the tongue was practically normal. The bladder control did not return to normal for another 4 weeks. The patient was discharged 6 weeks after admission.

This sixty-three-year-old undernourished, asthmatic woman developed pellagra ten days after the onset of pneumonia and five days after the temperature had returned to normal. She finally recovered from both the pellagra and the pneumonia.

Of considerable interest is the fact that she survived a second attack of pneumococcal pneumonia exactly two years after the one reported here. She was treated with sulfadiazine and large amounts of vitamin B complex from the onset, and experienced no complications.

Case 2. M. P., a 68-year-old edentulous widow, was seen because of an extremely sore mouth. One month previously she had had pneumonia with moderately severe jaundice. She had received no serum, and sulfonamide drugs had been withheld because of the jaundice. She ran a course characteristic of pneumonia and improved. The temperature became normal 16 days after the onset of the pneumonia. Twenty days after onset, she first complained of a sore mouth. A severe stomatitis was noted and sulfanilamide spray was prescribed. Three days later the mouth was full of so-called "Vincent's lesions," which were painted with 10 per cent chromic acid.

When I first saw the patient 12 days after the onset of stomatitis, the tongue was smooth, moist and fiery red along the edges. Toward the back there was a sharp line where the exfoliation had apparently stopped. There were two ulcers 3.5 cm. in diameter inside the right cheek and extending over the toothless lower gum. They were filled with blackish slough, the result of cauterization with chromic acid. The entire mouth and lips were extremely sore, and there were several small ulcers in both tonsillar regions and on the floor of the mouth. There was no dermatitis of the hands or legs, and no neuritis. A diagnosis of stomatitis due to acute vitamin B deficiency secondary to pneumonia was made, and the patient was started on 100 mg. of nicotinic acid and 4 brewer's yeast tablets three times a day. Local treatment was limited to a saline mouthwash.

Eleven days after the beginning of vitamin therapy the stomatitis had improved strikingly, although the left side of the throat was still sore. The ulcers inside the right cheek were one third their former size, and those in the tonsillar regions and on the floor of the mouth were clean and beginning to heal. The patient had some difficulty in mental concentration, some numbness of the right leg and soft and pitting edema of both legs up to the knees. At that time it was discovered that she had had peculiar dietary ideas for some years, having avoided all meats, drunk little milk and eaten only a few eggs for months, if not years, before the onset of pneumonia. Additional diagnoses of peripheral neuritis and probable protein deficiency were made, and the patient was urged to alter radically her eating habits.

When last seen 6 days later, the patient was feeling much better. The throat and mouth were no longer sore and she was eating well. She was having less difficulty with mental concentration, all the ulcers in the mouth were healed except one inside the right cheek, which was still 1 cm. in diameter, and the edema and the numbness of the right leg had disappeared.

This elderly edentulous woman with peculiar eating habits developed clinical pellagra, beriberi and probable protein deficiency following a moderately severe pneumonia. It seems likely that if the course of the pneumonia had been cut short by either drug or serum therapy, — jaundice complicating pneumonia is now known not to be a contraindication for either, — she might never have manifested outspoken signs of vitamin B or protein deficiency, in spite of her inadequate dietary and poor dental equipment.

Case 3. P. R., a 47-year-old, married woman of a slender, apprehensive type, was seen because of metrorrhagia. A uterine curettage 2 months later showed hypertrophic endometrium but no signs of malignancy. A hysterectomy was performed. Five days after operation the patient began to be nauseated and vomited several times. This continued for several days and the patient became extremely depressed. She had eaten almost nothing since the operation. The temperature had remained flat the first week after operation, never going above 99.6°F., but the pulse had slowly risen to 110. The patient was dehydrated, but the tongue aside from being dry appeared perfectly normal, being neither sore nor red. The possibility of vitamin deficiency and gastric dilatation was raised. Thiamin chloride and intravenous glucose and saline solutions were started and a tube was inserted in the stomach. One thousand cubic centimeters of black, foul-smelling liquid was removed and the patient felt better. The next day she had several loose stools and the tongue was still dry, so that more intravenous glucose and saline solutions were given. Another gastric lavage produced a large amount of green, foul-smelling fluid.

Nine days postoperatively, diarrhea was well established and the administration of 100 mg. of nicotinic acid every 4 hours was begun. On the 4th day, the patient looked better and the nausea, vomiting and diarrhea had stopped, but the mucous membranes of the entire inside of the mouth and tongue began to peel off. From time to time she was confused, irritable and difficult to handle, being extremely suspicious of visitors, nurses and doctors.

By the 12th day, the mental state seemed to be clearing, although the patient was still having three or four loose stools a day and occasionally vomited undigested food. By the 14th day she seemed mentally normal, the diarrhea had diminished to one or two loose stools a day, and vomiting had ceased. The temperature was normal and the pulse 90, and the oral mucous membranes were no longer fiery red or sore.

The patient again became psychotic on the 17th day, refusing all medication. On the 20th day, although progressing satisfactorily, she was still restless at night and developed an itching, erythematous eruption of both shins and the skin at the base of the neck. The lips were still dry and peeling and there was a small crack at the left commissure.

By the 26th day, the patient was practically well. The dermatitis, stomatitis, diarrhea, psychosis and tachycardia had all cleared. On close questioning it was found that during the 6 weeks between the time she was first informed that she would probably have to have an operation and its actual performance she had been so worried about the operation and its attendant risks and discomforts that she had eaten poorly.

This thin, anxious, forty-seven-year-old woman developed classic pellagra with diarrhea, dermatitis, stomatitis, psychotic manifestations and tachycardia during an afebrile postoperative course as the result of poor eating habits for six weeks prior to operation and practical starvation for the first

week after it, the deficiency of vitamin B being further accentuated by intravenous infusions of glucose solutions (cf. Cowgill's caloric vitamin B ratio[1]). One may speculate whether the tachycardia was due to thiamin deficiency although it is only of academic interest, since the patient obviously suffered from a niacin deficiency and there was almost certainly an associated thiamin deficiency.

Incidentally, it is also possible that vitamin B deficiency may be a contributory factor in some cases of postoperative gastric dilatation, intestinal distention and ileus and impaired bladder function.

CASE 4. H. C., a 69-year-old woman physician, was a chronic alcoholic and morphine addict. She had had so-called "colitis" for 10 years and had been diagnosed as having pellagra 5 months before I first saw her. She had been giving herself parenteral injections of a preparation of vitamin B complex since the diagnosis of pellagra had been made, but had continued her morphine and alcohol habits. She was seen in consultation because of cardiac failure. She was found to be obese, to have pinpoint pupils, — the result of morphine, — a severe glossitis, scaling and discoloration of the skin of the lower legs and roughness of the skin on the backs of the hands. The skin of both thighs showed innumerable scars of hypodermic injections. The heart was fibrillating (rate, 120), the blood pressure was 125/100, and there were signs of congestive failure. There were no murmurs, but the examination of the heart was not satisfactory owing to poor co-operation. The liver was felt two fingerbreadths below the costal margin. The reflexes were equal and active, and there was no tenderness on firm pressure on the calves.

Diagnoses of obesity, cardiac failure, morphinism, alcoholism and mild pellagra were made. The patient was rapidly digitalized and the previous dose of parenteral vitamin B complex was trebled. Three days later, when the heart rate was well controlled, she became completely disoriented. The daily morphine allowance was reduced, but she became psychotic and developed an intractable diarrhea. Although diarrhea is accepted as a common manifestation of withdrawal of morphine, there seemed to be an equal possibility that vitamin B deficiency was a contributory factor, so that 100 mg. of nicotinic acid was given by mouth every 2 hours. Two days later the patient was mentally clear, but the loose stools continued.

This obese sixty-nine-year-old woman, who was addicted to alcohol and morphine, had both severe cardiac failure and relatively mild pellagra. With adequate digitalization and gradual withdrawal of morphine she became psychotic and had severe diarrhea. The suggestion is made that one or both of these symptoms were manifestations of the pellagra, perhaps accentuated by the rapid restoration of cardiac compensation. One wonders whether some of the so-called "digitalis toxic psychoses" that appear in chronically decompensated cardiac patients, usually coincident with or immediately following a rapid diuresis and clearing in a few days' time with improvement of the cardiovascular status and ability to eat, may not be largely due to vitamin B deficiency.

Cowgill[11] showed that in dogs a diuresis produced by feeding large amounts of water by stomach tube hastens the time of onset of the anorexia characteristic of a deficiency of the vitamin B complex. His explanation is that significant amounts of vitamin B are carried off in the increased urinary output. If the concentration of vitamin B in edema fluid is significantly less than that in the circulating blood, a diuresis produced by digitalis or mercurial diuretics might well be expected to hasten the development of clinically apparent vitamin B deficiency.

CASE 5. S. C., a 57-year-old man, was seen at home complaining of pains in the legs and back and diarrhea of 2 months' duration. One year previously he had had intestinal obstruction, and at operation a carcinoma of the colon was found. The entire right colon was resected. He gained strength and felt extremely well for the next 10 months, but intestinal obstruction recurred and a second operation revealed extensive intra-abdominal and intrahepatic metastases. A short-circuiting operation was performed, but the patient had had diarrhea steadily ever since. He had also had considerable pain in the right lower quadrant of the abdomen and had taken a great deal of codeine in an attempt to control it. He had recently shifted to morphine by mouth, but was still having three to five stools a day and considerable aching in the legs and back. X-ray films taken the preceding week had shown no changes in the spine, pelvis or femurs. He had lost about 25 pounds in the preceding 2 months, but still weighed about 200 pounds.

Physical examination showed a well-developed and fairly well-nourished man who gave evidence of recent weight loss. The tongue and mouth were normal. There were multiple hard masses throughout the abdomen, and the liver was felt at the level of the umbilicus.

A week later, in addition to having diarrhea, the patient began to vomit two or three times a day. Two weeks after the first examination the tongue was noted to be fiery red and dry. The vomiting and diarrhea continued. Three days later the mouth became sore, and a week later it showed many shallow ulcerations. No attempt was made to give parenteral nicotinic acid, thiamin or vitamin B complex. It was impossible to determine how much of the pain was due to neuritis secondary to the vitamin B deficiency and how much to the known extensive carcinoma. Cure was impossible, so that only palliation was attempted in the last 2 weeks of life.

This fifty-seven-year-old man, who had extensive metastatic carcinoma of the liver and peritoneal cavity, a short-circuiting bowel operation and diarrhea of two months' duration, developed pellagra in the last two weeks of life. Profound malnutrition from chronic diarrhea, vomiting and extensive metastases in the liver were contributory factors.

CASE 6. A. F. M., a 72-year-old woman, was seen in 1936 because of an extremely sore tongue of at least 5 years' duration. Three years previously she had been minutely studied in a teaching hospital because of the same complaint. Moderate overweight, spoon-shaped fingernails and a smooth, shiny, red painful tongue were noted. Blood studies ruled out both pernicious anemia and hypochromic anemia, and no clue to the painful glossitis was discovered. All the teeth had been extracted some years previously, and double dentures had been worn ever since.

In 1936 the tongue still looked the same as it had in 1933, with the exception that there were some small superficial ulcerations at its tip. With the findings of Blankenhorn and Spies[12] in mind, a diagnosis of glossitis from vitamin B deficiency was made. A careful dietary history revealed a marked meat deficiency since the teeth had been extracted and a partiality for white bread.

During the next 2 years, tablets of brewer's yeast, nicotinic acid and riboflavin were administered in large numbers, with no apparent effect except a mild psychologic one. Finally, because of obvious failure to influence the painful affliction therapeutic attempts were abandoned for 4 years.

In 1942, the patient began to lose weight at an alarming rate. There were few symptoms except the earlier one of sore tongue, increasing difficulty with vision owing to cataracts, shortness of breath, and general weakness attributed

to arteriosclerotic heart disease. Extensive x-ray studies showed no evidence of organic disease other than arteriosclerosis.

The patient's own physician finally decided to try intramuscular injections of liver. The result was miraculous. Practically from the date of the first injection she began to improve. The first symptom to disappear was the sore tongue, which had been present almost continuously for 11 years. She next developed a ravenous appetite. In the ensuing 6 months she gained 20 pounds, and although she had not been expected to survive the winter, she became strong and well.

This elderly woman with double dentures had had a painful glossitis for eleven years that failed to respond to yeast, nicotinic acid and riboflavin by mouth but improved miraculously after injections of liver extract. Apparently the glossitis was of the type seen in sprue and pernicious anemia, which, as shown by Castle and his co-workers,[13] responds to liver extract orally or parenterally but not to orally administered components of the vitamin B group. Of considerable theoretical interest is the fact that even after eleven years the glossitis was still a reversible process and was completely relieved by the intramuscular injection of liver extract.

It seems not unlikely that a certain number of the symptoms usually associated with advanced age — mental confusion, loss of memory, disorientation at night, mildly paranoid ideas, asthenia, loss of appetite, weight loss and neuritis or rheumatic pains — are partially caused by vitamin B deficiency and not, as is usually assumed, solely by cerebral arteriosclerosis.

Case 7. H. D., a 38-year-old farmer and shipyard worker, with an unusually strong frame and a tremendous capacity for hard manual labor, had had pains in the forearms and hands for about 6 months, and cramps in the forearms sometimes kept him awake at night. Every morning it took him some time to limber up his fingers. Milking became extremely painful and using a hammer for more than an hour, as in shingling or caulking, became an impossibility. The hands and arms had never been frozen and the patient had not been exposed to lead paint or lead fumes for any period of time; he took alcohol only on rare occasions.

Physical examination showed a robust, red-cheeked extremely healthy-looking man weighing about 200 pounds. He carried 10 to 20 pounds of extra fat. The tongue was entirely normal, and the rest of the examination was negative. There was no demonstrable weakness of the muscles of the hands or forearms, and all the reflexes were normal.

Questioning as to diet revealed a special liking for potatoes, of which the patient ate six or eight a day, and white bread and a distaste for meat. His daily schedule involved getting up at 6 a.m., doing the milking and farm chores, working at a shipyard from 8 a.m. to 4 p.m., resuming his farm work, delivering milk and getting to bed between 11 p.m. and midnight.

On the remote chance that the pains were due to neuritis arising from a relative vitamin B deficiency caused by the enormous total metabolism, high-starch diet and low intake of vitamin B, 9 capsules of vitamin B complex were given daily by mouth and the patient was advised to take less potatoes and white bread. In 6 months the paresthesias had disappeared and the patient regained full use of his hands and arms.

This extremely vigorous, active, overweight farmer and shipyard worker, who averaged fourteen to eighteen hours of physical labor daily, developed a severe neuritis of both forearms and hands interfering seriously with his work. His diet was high in calories and starch and relatively low in foods containing vitamin B. His neuritis cleared up when his diet was corrected and supplements of vitamin B were added.

Obesity was possibly a minor contributory factor to the dietary deficiency in this case. It may contribute to vitamin B deficiency because it is always associated with an increase in total metabolism (Newburgh[14]), and because the more obese a person is the more likely his caloric vitamin B ratio (Cowgill[1]) is to be deficient. Adiposis dolorosa (Dercum's disease[15–18]), with its asthenia, neuritis and psychosis, sounds suspiciously like an extreme example of obesity predisposing to vitamin B deficiency.

Case 8. E. G., a 22-year-old stenographer, was seen with typical scarlet fever on the 3rd day of her illness. It had begun as an extremely sore throat. The patient had had great difficulty swallowing even fluids. The temperature was 105°F. and the pulse 130. The throat showed extensive exudate on both tonsils, and the right tonsil was pushed forward. There was a typical scarlatiniform eruption. The heart had no murmurs, and there was no history of rheumatic fever, chorea or a previous peritonsillar abscess.

The patient was hospitalized the next morning and was given antitoxin and sulfamerazine because she was extremely toxic. One day after admission the blood sulfamerazine level was zero, probably owing to an accidental confusion of blood samples, so that the drug was continued in full doses. The next day the level was 29 mg. per 100 cc. and the patient had oliguria. The drug was stopped and she was given glucose and saline infusions. Four days later the temperature fell to 100°F. Large quantities of urine were being excreted, but the patient had paranoid ideas, a gallop rhythm and what some observers called a pericardial friction rub but which I interpreted as a contact sound. At that time she looked a great deal better than she had when first seen 6 days before. The throat was much less sore, and she was able to swallow without much discomfort. The right tonsil was still pushed forward. There was fissuring at the outer corners of the eyes and lips. The tongue was smooth and red, with occasional enlarged papillas protruding above the surface. The heart was rapid and regular at a rate of 120/140, and the temperature was still 100°F. There was a well-marked gallop rhythm, and the above-mentioned scratchy sound was synchronous with both the 1st and 2nd sounds at the lower end of the sternum.

On the basis of paranoid ideas, cheilosis and embryocardia with gallop rhythm, a diagnosis of acute vitamin B deficiency was made. The contributory factors were sepsis, fever, inability to eat solid food for 8 days and intravenous infusions of glucose solution. A cardiac consultant made a diagnosis of acute rheumatic myocarditis and pericarditis, and the visiting physician a diagnosis of scarlet-fever heart.

The patient improved rapidly from this time on, having begun to eat well. The next day, she became and remained mentally clear, the heart began to slow down, and the friction rub disappeared almost at once.

Fifteen days after the onset of scarlet fever a peritonsillar abscess on the right ruptured spontaneously. At that time there were also migratory transient joint pains, with slight redness and limitation of motion for a few days. The electrocardiogram never showed a PR interval greater than 0.18 second, and all tracings showed a tendency to slight right-axis deviation. A soft apical systolic murmur developed, and the sedimentation rate was two or three times normal.

From the 17th day of the illness there were no further joint pains, fever or any other symptoms. The convalescence was uninterrupted. The sedimentation rate finally became normal 2 months after the first symptoms. When the patient was last seen, 10 weeks after the onset of the scarlet fever, the examination of the heart was entirely normal except for a faint apical systolic murmur, which was heard only when she was lying down and disappeared on exercise.

This healthy young woman with no previous history of rheumatic fever or any other type of heart disease developed paranoid ideas, cheilosis and tachycardia with gallop rhythm eight days after the onset of severe scarlet fever. The acute vitamin B deficiency was precipitated by fever, sepsis, inability to eat and intravenous infusions of glucose solution. It is impossible to establish an exact diagnosis of the cardiac lesion. Unfortunately, there are no pathognomonic signs or laboratory tests for differentiating or diagnosing some cases of rheumatic myocarditis, beriberi heart and scarlet-fever heart.

Wesselhoeft,[19] from his extensive experience with scarlet fever, describes scarlet-fever heart as a transient affair lasting for a few days to three weeks. It usually consists in the development of murmurs, without irregularity in rhythm. The first sign is generally a split first sound. Abnormalities are likely to be noticed when the patient is first allowed out of bed. The murmur is extremely variable in time and character. It may occur in systole or diastole, but more frequently in the former. Some writers have emphasized that its occasional harshness is somewhat suggestive of a slight friction rub at the base. The heart rate is not necessarily increased with the advent of these abnormal sounds, but quite independently of them there may occur a persistent tachycardia or even a relative bradycardia. There is rarely any cardiac enlargement, and there is no precordial pain or discomfort. All these clinical signs usually disappear by the end of a week, and in marked cases in three or four weeks. Electrocardiographic changes are variable, inconstant and nonspecific.

A similarity may be seen in this description between scarlet-fever heart and the milder forms of beriberi heart disease (Weiss and Wilkins[8]). There are no clinical or laboratory tests that distinguish between them with certainty. The suggestion is made that they may be identical conditions. The excellent prognosis of practically all cases of scarlet-fever heart may be explained by the fact that once the acute sore throat of scarlet fever subsides the patient begins to eat. Also, the normal nutritional status of most persons preceding an attack of scarlet fever renders them less likely to develop the more advanced stages of beriberi heart disease.

Conclusions

The above 8 cases of clinical vitamin B deficiency were seen in seven years of private practice. They have been reported in the hope of emphasizing the frequency with which this group of diseases occurs as a complication of many serious illnesses seen every day in practice. Therapy is self-evident if the diagnosis is made. An acute awareness of the existence of such conditions is necessary if these cases are to be correctly diagnosed and treated in their earlier stages.

The precipitating or contributory factors in this small series were fever, a bizarre diet, chronic diarrhea, poor teeth or poorly fitting dentures, preoperative apprehension, infusions of glucose solution, hard manual labor, obesity, drug addiction, diets rich in starch and sore throat or sore mouth. In any given case there was usually more than one factor.

It may be repeated that symptoms of vitamin B deficiency may develop whenever an elevated metabolism occurs simultaneously with a deficiency of vitamin B-containing substances in the diet or any interference with absorption from the gastrointestinal tract. These should be looked for in cases of fever, increased caloric intake, increased muscular activity, hyperthyroidism, chronic diarrhea or vomiting and the like, with or without obvious limitation of intake or absorption of vitamin B-containing foods.

51 Brattle Street

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THE PURPOSE AND ACCOMPLISHMENTS OF THE LAWRENCE CLINIC

H. FRANK McCARTHY, M.D.,* AND FREDRICK C. ATKINSON, M.D.†

LAWRENCE, MASSACHUSETTS

IN 1936, one of us (H.F.M.) became convinced, through observations made during the course of his practice, that there should be in Lawrence an institution devoted primarily to the care of the medically indigent. It was noted that because of the lack of adequate outpatient facilities, poor patients were prone to employ a physician or surgeon until their accumulated bills became prohibitive, and then, because of inability to pay the incurred debt, "shopped" from physician to physician. Because of numerous experiences of this sort, it was obvious that there was an urgent need for this charitable institution. Accordingly, the interest of the late Dr. W. Dacre Walker, a physician of influence and high repute in this community, was enlisted, and after considerable study and thought he gave the project his wholehearted support. Dr. Walker became the first president of the Board of Trustees and the first chief of staff.

On November 13, 1936, the charter was granted and a group of outstanding lay persons consented to act as trustees. A meeting was held by nine representative physicians and dentists of the community and plans to open a free outpatient clinic were formulated. Aided by funds and equipment donated by these physicians and dentists, on January 6, 1937, the clinic made its inauspicious start, poorly housed in a somewhat dilapidated local building. A certain amount of criticism, which is usually attendant on an innovation, was at first directed toward the clinic, owing to the mistaken impression that its primary purpose was the financial gain of its sponsors. Criticism abated and suspicions were allayed once the public became convinced of the worthiness and sincerity of the project.

It was early recognized that the services of the clinic would be sought by many who could afford private care. Accordingly, rigid rules for admission were devised. It was decided that candidates for the clinic should be admitted only on the presentation of a reference from a physician, dentist, minister or recognized charitable agency, stating that the bearer could not afford to pay for private care. In a few cases it was found that the referrer had recommended patients who were not financially deserving of clinic care. They were warned, and if the offense was repeated their privilege to refer patients was revoked. Again, on a few occasions it was found that physicians referred patients for special work, such as laboratory or x-ray examination, who were not eligible, but when it was explained to them that patients must also be of clinical status to receive this care, the difficulty was eliminated.

SOCIAL RELATIONS

The case load in the early days of the clinic was extremely heavy because of conditions in the mills following the Depression. Recently, with the advent of war production, the load has dropped considerably and, for the first time in many years, people who were formerly on the relief rolls and had received free care at the clinic have become contributors to its support. This point is important, since in a community of the type of Lawrence the borderline between prosperity and poverty is frequently the holding of a job. In the files of the clinic are hundreds of letters of gratitude written by former patients who wish it to be recognized that they have now assumed the role of givers rather than receivers.

The clinic is at present maintained by public donations, which are solicited by mail. About $10,000 is received yearly from approximately 3000 donors. There are a few large annual donations made from several organizations, but it is apparent from these figures that the bulk of the money received is in small amounts and is sent from a cross section of the working people of the community. A survey of the list of donors is convincing proof that the clinic is being largely supported by potential patients, and a great many of their letters indicate that they are cognizant of this fact.

STAFF

The original charter members interested other members of the medical profession in the project. These physicians and dentists staffed the various departments and have given generously of their time year after year, with no remuneration other than the gratitude of the patients. Many of the original staff were young men, not too busy in their private practice and not at the time affiliated with any other local institution, so that they could afford to spend considerable time when the medical case studies were to their professional advantage. For the cases that require special study, ten outstanding Boston specialists act as consultants to the various departments, and the clinic has been accorded the privilege of referring to them such patients as require their attention. These men, also, have worked without financial remuneration.

As in other instances, the war has brought special problems to this institution. As stated above, the clinic was originally staffed almost wholly by youthful physicians and dentists. Now that the various

*Chief-of-staff, Lawrence Clinic.
†Head of Medical Department, Lawrence Clinic.

branches of the service have recruited most of them, out of the original staff of fifty-two only nine are now active at the clinic. These men, however, can adequately handle the work because of the lighter case load resulting from improved economic conditions.

Administration

The number of paid workers at the clinic is eight, consisting of a superintendent, a secretary-bookkeeper, a clerk, a laboratory technician, a medical assistant, a registered nurse, a dental hygienist and a janitor. These paid workers, however, are augmented by numerous volunteer workers in all departments from office work to medical assistance. Most of the administrative staff are underpaid in comparison to their efforts, but have stayed on in spite of opportunities for higher pay, feeling recompensed by the satisfaction that comes to those who render a community service. Through the kindness of a prominent local accountant, a bookkeeping system was installed, and this is audited yearly without charge.

Facilities

In April, 1940, after three years of operation in humble surroundings, the clinic moved to its present location. Under the supervision of the Board of Trustees, the building in which it is now housed was purchased, and although heavily mortgaged at the time of purchase, it was possible to renovate it completely and to equip each department with the most modern equipment. The rooms, which are clean and wholesome, are unpretentious and strictly utilitarian. Most of the available money was expended on good medical equipment rather than in beautifying the surroundings. This, in the opinion of the staff, was much more advantageous to the patients' welfare. All departments are well equipped, and they include a dental clinic with two chairs. Some of the departments are supported by various organizations, as in the case of the Eye, Ear, Nose and Throat Department, which is sponsored by the local Lions Club.

Medicine is dispensed free or at cost. In 75 per cent of the cases it is given without charge. Appliances such as arch supports, eyeglasses and back braces are obtained for patients at cost, but if the latter are unable to pay even this amount, an endeavor is made to procure the appliances through some welfare organization. As a last resort, the clinic will purchase such appliances for a worthy patient.

When a patient needs hospitalization for surgery, efforts are made to secure a free bed in a private hospital, and the clinic's staff surgeons do the operating without charge. In certain cases patients are referred to the municipal hospital.

Among other facilities available to patients is a completely equipped x-ray department. Here also, the patient if financially able pays the exact cost of the material used. If he is unable to pay, the work is done free. The clinic has a well-equipped laboratory with a competent technician in charge.

In June, 1938, a state venereal-disease clinic was assigned to the Lawrence Clinic. Needless to remark, this is always one of the busiest departments. The State reimburses the clinic on a per capita basis for each patient treated, and although no profit results from this arrangement, it makes this particular department self-supporting.

On first admission, a patient is requested to pay an admittance fee of 50 cents, and 25 cents for each successive visit. The admission fee was decided on as a morale factor and for its psychologic effect on the patient rather than for the financial aid expected from it. About 65 per cent of the patients are unable to pay even this small fee and are admitted free. Their ability to pay or to receive free care is determined on the first admission by the admitting clerk. The economic status of each patient is watched as carefully as possible, and on improvement in such status, the patient is urged to return to his family physician for medical care until he again becomes eligible for clinic care.

Ladies' Auxiliary

Shortly after the opening of the clinic, many civic-minded women volunteered their services to the institution in various ways. They often drove elderly patients to and from their homes, made medical supplies, sewed curtains and in many other ways assisted in the undertaking. This group was the nucleus of a larger group of women who formed the Ladies' Auxiliary. This auxiliary has been invaluable to the clinic. Its members have sponsored benefit parties and have raised considerable sums of money. They have been to a great extent a factor in promulgating goodwill throughout the community and in helping to dissipate any latent suspicions that might still be entertained regarding the purpose of the institution.

* * *

The clinic is about to enter on its seventh year of service to the medically indigent of Lawrence and the surrounding communities. During the past years approximately 100,000 treatments have been received by these people. If it is borne in mind that all the patients have been bona-fide charity patients, it will be realized that the institution has justified its existence and established its position as a social necessity. If in this period only a few human beings had been rehabilitated, — and there have been hundreds, — the sponsors of the clinic would still feel that the end had been worth the means.

MEDICAL PROGRESS

PIGMENTATION OF THE SKIN (Concluded)*

HAROLD JEGHERS, M.D.†

BOSTON

MELANOSIS ASSOCIATED WITH AN INCREASED NUMBER OF MELANOBLASTS

BECKER and Obermayer[102] classify a number of pigmentary disturbances as melanosis associated with an increased number of melanoblasts (Table 2). Some of these are of general medical interest. The increased melanoblasts of the blue nevus (see colored plate[305]) and of Mongolian spots (see colored plate[28]) are deep in the corium, so that the resulting melanin pigmentation appears blue. Blue nevi bear the same relation to Mongolian spots as the ordinary brown moles do to brown-pigmented skin.[306] Mongolian spots are frequently noted over sacral and other areas in the darker races and occasionally in the white-skinned races, and persist for a variable number of years after birth.[27]

Ephelides (freckles)[11, 22, 102, 103] are due to increased aggregates of melanoblasts producing accentuated, sharply demarcated yellowish-brown areas, often zizzag in outline. They are more obvious in spring and summer, can be seen with Wood's light when invisible to the naked eye (see illustration[307]), appear early in life but not in infancy, never occur on the palms and soles and are prominent on the exposed parts of the body, and the predisposition to them is an inherited dominant characteristic. They may be confused with lentigo and melanosis produced by disease.

Melanotic neoplasms of the skin are frequently encountered. The difficulty of treating them and the frequency of metastasis from them are well known. Of significance here is the observation that aside from true metastases, malignant melanoma may cause a diffuse and marked melanin pigmentation of the skin.[308-310] In a reported case (see illustration[308]) a white man became so dark in color that he was thought to be a Negro.

Sharply outlined brown blotches, *café au lait* spots and large areas of brown skin pigmentation (see illustrations[311,312]) are among the commonest features of neurofibromatosis (Recklinghausen's disease), and in some reported series were noted in every patient.[311, 313] The pigmented spots are usually not congenital and may develop in childhood or later in life. The pigmentation in this disorder is considered to be due to a localized anatomic anomaly in the cutaneous nerve itself that produces the increase of melanin in the epidermis.[313] In its classic form neurofibromatosis is readily recognized. It is, however, not well appreciated that in the formes frustes[312, 313] pigmented spots on the skin may be the only visible manifestation of the disorder. It is now well known that neurofibromatosis may involve many internal organs, including the bone, the central nervous system, and the endocrine organs, and it is therefore of general medical interest.[312, 314] The possibility of neurofibromatosis should not be overlooked in persons with patches of brown skin pigmentation of this type.

Much interest has been aroused in recent years in a curious syndrome characterized by osteitis fibrosa disseminata, areas of pigmentation and a gonadal dysfunction, especially precocious puberty in the female.[315, 316] It has been referred to in the recent literature as osteitis fibrosa cystica (Albright) and Albright's syndrome. The disorder is not due to parathyroid disease. The patchy skin pigmentation seen in this disorder is almost constantly present, is brown in color with no change in skin texture, is due to melanin, and on histologic examination is similar to the pigmentary lesion of neurofibromatosis (see illustrations[315]). The skin pigment is often distributed in the same general area of the body as that in which the cystic bones are located. Its occurrence in children, the pigmented areas and endocrine stigmas, lack of generalized decalcification and normal blood calcium and phosphorus values serve to distinguish this disorder from hyperparathyroidism with osteitis fibrosa cystica generalisata. Thannhauser[313] has recently suggested, citing much evidence in support of his belief, that the pathogenesis of neurofibromatosis and that of osteitis fibrosa cystica localisata and disseminata are related. He refers to them as neurofibromatosis (Recklinghausen) and osteitis fibrosa cystica localisata et disseminata (Recklinghausen). The many similarities of the pigmentary syndromes of both conditions are strikingly emphasized in his paper.

Leschke's syndrome,[317] the association of the pigmentary syndrome of neurofibromatosis with an endocrine syndrome, is probably only a variant of neurofibromatosis. It has been especially emphasized in the German literature (see illustration[318]).

The association of skin melanomatosis with melanomatosis of the central nervous system, aside from the possibility of a skin melanoma metastasizing there, has been reported [319, 320] and is considered

*From the Evans Memorial, Massachusetts Memorials Hospitals; the Fifth and Sixth (Boston University) Medical Services, Boston City Hospital; and the Department of Medicine, Boston University School of Medicine.

†Associate professor of medicine, Boston University School of Medicine; physician-in-chief, Fifth Medical Service, Boston City Hospital, and assistant physician, Clinical Staff, Evans Memorial, Massachusetts Memorial Hospitals.

a congenital neurocutaneous syndrome and grouped with tuberous sclerosis of Bourneville and Brissand, neurofibromatosis and hemangiomas of the meninges associated with similar nevi of the skin. The pigmented spot that occasionally occurs on the back over the site of a spina bifida occulta probably also belongs in this group of pigmentation due to increased melanoblasts.

Hemoglobin Pigmentations

Variations in skin color produced by changes in the amount or nature of the hemoglobin contained in the capillaries and subpapillary plexuses of the skin are far more frequent in medical practice than are the types due to deposition of an exogenous or endogenous pigment in the tissues of the skin. This review does not cover them in any detail, but the more important ones will be mentioned so that the wide range of possible skin color change will not be overlooked.

Environmental and emotional control of skin capillaries may cause a red-flushed, a pale or a cyanotic skin. Pallor due to anemia, the reddish-blue facies of polycythemia vera (see colored plate[321]), the cherry-red color of carboxyhemoglobinemia (see colored plate[322]), the bright red color of nitricoxide-hemoglobinemia which may result from poisoning by fumes from combustion of explosives, the chocolate-blue color of methemoglobinemia (see colored illustration[323]) and the lead or mauve-blue color of sulfhemoglobinemia are striking and contrasting examples. The color of cyanosis due to increased reduced hemoglobin may vary from the deep purplish blue (see colored plate[324]) of strangulation to the heliotrope cyanosis (see colored plate[325]) noted in certain types of pneumonia. Lewis's[3] colored plates for the study of cyanosis illustrate the many tones and hues that the skin color may manifest in this condition. A predominance of oxyhemoglobin in the skin color accounts for the redness of such conditions as the flushed face of alcoholism, the emotional blush, erythemas and inflammatory lesions. Hydrocyanic acid combines poorly with hemoglobin or oxyhemoglobin, but combines readily with methemoglobin to form cyanhemoglobin, which differs from ordinary hemoglobin in its bright-red color. This compound is responsible for the bright ecchymotic spots often seen in the skin of patients who have died from hydrocyanic poisoning.[326] Pronounced melanin pigmentation of the skin may serve to screen out influence on skin color due to hemoglobin variations, — that is, in Negroes, — but this is not generally so marked for mucous membranes.

Metallic Pigmentations of the Skin

Hemosiderosis

"Hemosiderin" is the name applied to an iron-containing pigment that mainly occurs in the body as a result of local or general destruction of blood and in hemochromatosis. "Hemosiderosis" is a general term used to designate the process characterized by deposition of hemosiderin pigment in the superficial corium, with resultant pigmentation. How much of the pigmentation can be directly attributed to hemosiderin deposition alone and how much to melanin production initiated through some local action or through deposition of hemosiderin in the adrenal glands is not entirely clear. In some forms of hemosiderosis an accompanying melanin pigmentation — that is, hemochromatosis — is common. Hemochromatosis and a variety of hematologic disorders are the main systemic causes of hemosiderin formation. The most frequent types, however, occur on the lower legs because of a variety of combinations of local and systemic disturbances.

Hemochromatosis. Pigmentation of the skin is one of the most constant and characteristic features of hemochromatosis. Butt and Wilder[327] noted its absence in only 1 of 30 cases studied by them. Case reports stressing the rarity of its absence attest to the almost constant presence of the pigmentation.[328, 329] This may appear late in the disease, but it is often present for years and is frequently the first symptom to attract attention.[330]

A detailed discussion of skin pigmentation is given by Sheldon[330] and Hellier.[331] Hemofuscin, an iron-free pigment, although usually present in hemochromatosis is not highly important in causing skin pigmentation. Melanin and hemosiderin are the two pigments concerned with the changes in color of the skin. Hemosiderin is found chiefly in the corium, mainly in the neighborhood of the sweat glands, and perhaps to some degree in the deeper epidermis.[330] It is at times solely responsible for pigmentation of a bluish-black, slate or lead color, which is sometimes generalized but is always more marked on the face, the neck, the extensor surfaces of the limbs and usually the external genitalia.[330, 331] Melanin is found in the deepest layers of epidermis and occasionally to a small degree in the chromatophores of the corium. Alone, it causes the usual brownish type of pigmentation characteristic of melanin pigmentation.

It appears from case reports that melanin tends to be the earlier type of pigmentation. This is suggested by the almost constant presence of hemosiderin in the skin in autopsy reports, whereas many biopsy studies of the pigmented skin at earlier stages reveal no hemosiderin. When both pigments are present, melanin modifies the color and blends the slate gray of hemosiderin with brown to give a bronzed appearance[331] (see colored plates[332–335]).

Hellier[331] records the findings of the skin histology of 57 cases from the literature and his own as follows: hemosiderin was present in 48 cases, absent in 5, and not mentioned in 4; melanin was present in 35 cases, absent in 12, and not mentioned in 10.

In 8 cases hemosiderin was present without melanin; 7 of these patients had a slate-gray or bluish color. It is evident that melanin pigmentation is far more frequent and contributes more to the skin color than is generally appreciated. The classic bronze color may depend on this frequent combination of the two pigments. The early appearance in the skin of melanin pigmentation alone suggests that the mechanism is related either to hemosiderin deposition in the adrenal glands or perhaps to cortical dysfunction of some other nature.[330, 331]

In Sheldon's[330] extensive review the mucous membranes were reported as pigmented in 16.7 per cent of the cases studied — usually in the mouth and next most frequently in the conjunctivas. The pigment was apparently of the melanin type, although one report of the demonstration of hemosiderin was given.

The intensity of the pigmentation has been reported as occasionally increasing noticeably shortly before death,[330] and also as diminishing rapidly when the diabetes accompanying the disorder is controlled with insulin.[336] This is usually attributed to a metabolic change within the skin cells that alters its optical character rather than to dissolution or removal of the pigment.[330]

Skin biopsy in hemochromatosis is often poorly handled for the best results. The pigments are diffusely deposited in the skin over the entire body. One should avoid taking the skin from the legs, over pressure areas or irritated areas or from a body fold. Hemorrhagic disorders and blood dyscrasia as causes of hemosiderin formation must be excluded by appropriate hematologic studies. A request should be made for the study of melanin pigmentation as well as of stains for the iron-pigment fraction. Other aids to the diagnosis of hemochromatosis are Rous's test of the urine sediment,[337] examination of sediment from ascitic fluid for iron pigment,[338] the presence of insulin-resistant diabetes[339] or, for that matter, any degree of diabetes in a pigmented patient or diabetes with an elevated basal metabolic rate, pigmentation in a patient with ascites, cirrhosis or an enlarged liver, Fishback's skin test[340] and evidence of sexual hypoplasia.[341]

Hemosiderosis of the lower legs. Hemosiderosis of the lower legs with skin pigmentation is frequently encountered in both general and dermatologic practice. Either gross or microscopic extravasation of red cells into the skin for long periods of time leads to deposition of hemosiderin pigment in the corium and gradually to pigmentation of the skin, usually a brownish, sepia or copper hue. It may be fairly diffuse or centered about ulcers of all types. It is oftenest seen with the stasis of varicose veins but is not uncommon in patients with other types of stasis, for example, chronic ascites.

Inflammatory and other stimulants for melanin formation are also present. Patients with this type of leg pigmentation who have glycosuria, an enlarged liver, cirrhosis, ascites and so forth are often erroneously suspected of having hemochromatosis and a biopsy specimen of skin is taken from the pigmented area, the report of which leads to more confusion.

Supporting pressure on the tissues from shoes tends to protect the feet from the hemosiderosis. It is likely to commence near the ankle, to extend a variable distance toward the knee and to be more prominent anteriorly than posteriorly. Any hemorrhagic disorder if chronic, increased fragility of capillaries, diminished tissue elasticity of old age and so forth, if added to venous stasis, may accentuate the development of the process.

Of general medical interest is the occurrence of this type of skin pigmentation as a sequela of repeated mild trauma to the lower legs, in stasis from varicosities and probably other causes, in hemochromatosis (here diffuse over the body and not limited to the legs); about the leg ulcers in sickle-cell anemia,[174] congenital hemolytic jaundice,[171, 172] in Cushing's disease,[370] Gaucher's disease,[185] in pernio,[342] and probably in other conditions. The occurrence of pigmentation on the lower legs, especially in a young person without varicose veins, may be a clinical finding of some significance. Of dermatologic interest are Schamberg's disease[343, 344] (see colored plate[345]), Majocchi's disease[344] and others.[343, 346, 347] These disorders are listed in Table 3.

TABLE 3. *Causes of Hemosiderosis of the Lower Legs.*

CAUSES OF GENERAL MEDICAL INTEREST	CAUSES OF DERMATOLOGIC INTEREST
Repeated minor trauma	Schamberg's disease
Venous stasis of leg veins, especially from varicose veins	Majocchi's disease
Hemochromatosis	Pigmented purpuric lichenoid dermatitis
Gaucher's disease	
Cushing's syndrome	
Pernio*	
Congenital hemolytic jaundice*	
Sickle-cell anemia*	

*Included in this tabulation on circumstantial evidence. The nature of the pigmentation is not clearly stated in the available literature.

Argyria

Argyria is the pigmentation resulting from deposition of silver in the corium of the skin and mucous membranes. The monograph on argyria by Hill and Pillsbury[348] based on over six hundred references attests to the world-wide interest in this subject. Any silver compound is capable of producing argyria when used for a sufficiently long period of time by any route of administration other than the intact skin. Argyria may result from the therapeutic use of silver as well as industrial exposure to it.[349] The incidence of the condition appears to be increasing.[348, 350] Silver is accumulated in the body over many years, and argyria develops when a sufficient amount has been deposited in the skin. The last silver preparation used before the

appearance of argyria is often erroneously blamed; if not sufficiently large in itself, it was only the amount that, added to previously stored silver, allowed the visual threshold to be reached. Silver can be demonstrated quantitatively in the skin by spectroscopic examination long before it is clinically observable.

Argyria is permanent and, other than the cosmetic disfiguration, has no effect on health. No adequate method of treatment is available, but small areas may be partially bleached.[351] Silver is deposited uniformly over all skin and mucous membrane surfaces in the corium but not in the epithelium. The areas of the body exposed to sunlight become strikingly accentuated, and such exposure should be strictly avoided by those with argyria of even minimal degree. Histologic diagnosis can readily be made from a skin biopsy.[352]

Argyria is clinically most pronounced on the face, hands, conjunctivas and fingernails. Because of pigment deposition in the corium, the color of argyria is usually described as slate gray, blue gray or lead, or in other words a blue varying from gray to blue (see colored illustration[349]). The ability of the pigmentation to simulate cyanosis is striking.[348, 349] Levine and Smith[353] recently reported 5 cases of argyria erroneously treated for cardiac disease because the argyria pigmentation had been mistaken for cyanosis. This has occurred in 3 patients studied at the Boston City Hospital. If the pigmentation is predominantly of a lead rather than a blue hue, the condition may be confused with melanosis or hemochromatosis.

Chrysiasis

Schmidt[354, 355] has presented a valuable review of chrysiasis. He defines it as "a permanent pigmentation of the skin caused by the parenteral use of a gold preparation and the subsequent exposure of the skin to ultraviolet radiation, including sunlight." This potential hazard should be known to all physicians using gold. Chrysiasis is not uncommon after its excessive use.[354] A latent period of one month to five years may elapse before the skin pigmentation makes its appearance; it then persists permanently. Lorenzen's[354] study showed that "no patient receiving a total dose of less than 50 mg. of gold sodium thiosulfate per kilogram of body weight had definite chrysiasis and that no patient escaped having the condition if the total dose received was 150 mg. per kilogram of body weight." Cases in the earlier literature were noted after the gold therapy of tuberculosis, but gold therapy has been extensively used in arthritis in recent years, apparently without causing chrysiasis.[356] The disease may appear only after a long latent period, so that a long follow-up is needed for one to be certain on this point.

The involved skin has been described as uniformly gray, slate gray, grayish purple, grayish blue, bluish violet, blue green and brownish yellow.[354] Chrysiasis appears after exposure to sunlight and is usually limited to the exposed portions of the body — the face, neck, arms, hands and scleras. The mucosa of the mouth is usually not stained. Predominant deposition in the upper corium accounts for the characteristic gray to blue hue of the skin pigmentation. Diagnosis can be made from dark-field study of histologic sections.[348, 357] Treatment is not successful. Prophylaxis depends on a minimal dosage of gold salts and avoidance of undue exposure to sunlight or ultraviolet radiation.

Bismuthia

The prolonged oral use of bismuth has been reported as rarely producing a generalized persistent skin discoloration (see colored plate[358]) resembling argyria that has been called "bismuthia." The conjunctivas and oral mucosa are pigmented. Bismuth is identified by chemical examination. The pigment granules are seen on histologic study to be evenly scattered through the corium. The rarity of this condition in spite of the common use of bismuth is worthy of emphasis. The factors essential for its production are not known. A black line of bismuth sulfide along the gums near the teeth is frequently seen in persons treated with bismuth.[359] Tartar deposits on the teeth accentuate its formation. Pigmentation of the tongue and buccal mucosa is a warning symptom of impending stomatitis and nephritis.[360]

Wiener[360] reported a case of bismuth pigmentation of the entire vagina in a syphilitic woman with ulcerative cervical cancer who had received bismuth therapy. This clinical syndrome appears at first glance to be analogous to the bismuth pigmentation of the gums, but may be more complex. Sulman et al.[361] treated 122 female albino rats with daily injections of bismuth and estrogenic or gonadotropic hormone preparations. Thirty to 80 per cent of the animals showed bismuth pigmentation of the vagina, but 60 control female rats treated with bismuth alone showed no blackening of the vagina. The pigmentation was believed to have been conditioned by hyperemia of the vagina from the hormone therapy, together with some unknown factor causing selective fixation of bismuth in the vagina.

Hydrargyria

Face creams containing mercury or mercury and bismuth may produce a peculiar discoloration of the skin (a brownish, gray or slate color), usually of the face and neck and limited to the areas where the material is applied, with accentuation in skin folds (see illustration[362]). Biopsy material shows mercurial deposits in the epithelium and corium.[362–364] This type of pigmentation is purely of local origin. Goeckermann[364] has commented on the ease with which it has been confused with pigmentations of

systemic origin. The differential diagnosis of facial pigmentations is given in detail by Hollander[362] and others.[289, 365]

Lead Pigmentation

The deposition of lead as lead sulfide within the tissue of the gums about 1 mm. from the border of the teeth, producing the so-called "lead line" or "Burtonian line" is widely recognized as one of the characteristic but not constant signs of lead poisoning. Lead does not cause pigmentation of the skin. The ashen-lead color of the face and pallor of the lips noted in lead poisoning is believed to be due not only to anemia but in addition to a possible action of lead on the skin capillaries.[366, 367]

Iron Pigmentation

A permanent tattoo type of brown pigmentation of the skin may result if ferrous sulfate or ferric chloride solution along with solution of lead or aluminum acetate is used on a vesicular or open skin lesion, allowing contact of the chemical with the corium. Pigmentation is due to dyeing of the corium with basic ferric acetate. Other agents than acetate may likewise enable iron to fix itself permanently to the protein molecule.[368–370] This type of pigmentation is purely of local origin. In contrast to hemosiderin, metallic iron does not cause pigmentation of the skin through systemic action.

Miscellaneous Pigmentations

Porphyria

Watson[371] in reviewing the clinical features of porphyria points out that pigmentation of the skin is not uncommon in this disorder but that the nature of the pigment has not yet been determined. In congenital porphyria, he adds, uroporphyrin has been identified in the epithelial cells, where it is undoubtedly responsible, at least in part, for the marked sensitivity to light, a phenomenon that is usually absent in the intermittent acute type. In the congenital form of porphyria the teeth may be stained pink (see colored plate[372]).

Pigmentation of the skin in the intermittent acute types of porphyria is much more prominent on the exposed surfaces than on others; it may be patchy or diffuse, but at times covers the entire body and is bronze. Watson[373] has not noted pigmentation of the mouth or other mucous membranes, a point also made by Nesbitt.[374] Vitiligo was noted in 1 patient,[371] an observation suggesting a disturbance of melanogenesis in the skin. Whether the responsible pigment is melanin is not known. Porphobilin, a red-brown pigment occurring in porphyria, somewhat similar to urobilin but not having porphyrin characteristics, may be related to the skin color.[371] It would be quite compatible with its physical characteristics to find deposits of it in the skin.[371] Turner and Obermayer[375] report a case of porphyria accompanied by epidermolysis bullosa, hypertrichosis and melanosis.

Methemalbuminemia

Fairley[169,170] has recently given a detailed account of the blood pigment methemalbumin, first described by him in 1934. The chocolate-brown color it gives to plasma and its absorption characteristic on spectral analysis are so similar to the effects of methemoglobin that they have in the past been readily confused. It is pointed out that both methemoglobin and sulfhemoglobin are essentially confined within the red cells and that the term "methemoglobincythemia" should replace "methemoglobinemia" and "sulfhemoglobincythemia" should replace "sulfhemoglobinemia." Methemalbumin, on the other hand, is found only in the plasma. Hematin cannot exist as such in the circulating blood, and when formed combines with serum albumin to form methemalbumin. Methemalbumin is responsible for the brown color of plasma in blackwater fever and may be present in variable amounts in other hemolytic anemias. How much influence this pigment has clinically in causing changes in skin color is not clearly stated in the literature.

Malarial Pigmentation

Books on differential diagnosis commonly list chronic malaria among the disorders causing skin pigmentation. Some melanin pigmentation no doubt occurs, but the pigment problem in malaria is by no means simple. It is well known that the malarial pigment hematin is widely deposited in tissues, particularly in those of certain visceral organs. Furthermore, hemosiderin is formed.[375] Jaundice occurs in some forms of malaria. Hemoglobinemia and methemalbuminemia occur in blackwater fever.[169, 375] Strong's[376] monograph on tropical medicine gives a detailed discussion of this complex subject.

Pigmentation Due to Other Medicinal Agents

Many colored chemicals, used either therapeutically or diagnostically or acting as poisons, often produce temporary skin discoloration. All physicians are familiar with some of these substances. Typical of this group are methylene blue, injectable Prontosil, Evans blue, Congo red, acriflavine, santonin, atabrine, dinitrophenol and many others.

Other Pigmentations

Vitiligo and gray hair. Vitiligo and canities represent diminution or absence of normal pigmentation of the skin and hair, respectively. Vitiligo stands in strong contrast to the hyperpigmentation of the skin discussed in this paper. It is beyond the scope of this review to discuss these subjects in detail. Considerable interest now centers in the possibility that the presence of gray hair is in some way related to a deficiency of one or more of the factors

of the vitamin B complex. An appraisal of the therapeutic efficacy of vitamin treatment of gray hair is not warranted at present. The status of this subject is given in two recent reviews.[377, 378] Undoubtedly there is much to be learned about the systemic causes and associations of gray hair, as witness its occurrence in such widely separated disorders as pernicious anemia, Werner's syndrome[208] and the Vogt–Koyanogi syndrome.[379]

It is of considerable interest that patches of vitiligo not uncommonly coexist in the very skin showing hyperpigmentation. Vitiligo occurs in Addison's disease, hyperthyroidism, scleroderma, arsenic poisoning, pernicious anemia and many of the other diseases with melanin skin pigmentation. These paradoxical combinations at present admit of no clear-cut explanation. Vitiligo may result from a variety of local skin lesions, a subject well discussed in the standard textbooks on dermatology. Vitiligo also frequently occurs without any apparent local or systemic cause.

It is also of interest that Mussio Fournier, Cervino and Conti[380] have reported using pituitary melanophore hormone preparation for the treatment of vitiligo, apparently with some success, whether this preparation was given intradermally, locally by electrical transfer or parenterally.

Pigmentation of mucous membranes of mouth. The value and limitations of pigmentation of the mucous membranes of the mouth as a diagnostic sign of systemic disease appear to be poorly understood. Many physicians associate it exclusively with Addison's disease. The literature contains a number of excellent discussions of this subject.[381–385]

Oral pigmentation (melanoplakia) is normally a racial peculiarity. Monash[383] demonstrated that only 5 per cent of 200 Negroes showed no pigmented areas in the mouth. During a six-month period he noted oral pigmentation in 30 persons of various racial groups including Italians, Jews, Greeks, Roumanians, Chinese, Syrians and others. He gives an excellent description of physiologic oral pigmentation, which is minimal in infancy and complete by the end of the second decade. Prinz and Greenbaum[381] discuss in some detail racial, physiologic oral pigmentation. The work of Becker,[20] Laidlaw[386] and others showed that in the majority of white persons pigment cells (melanoblasts) can be demonstrated microscopically in the oral mucosa even if pigmentation is not grossly observable. These pigment cells are therefore subject to many of the same factors known to influence similar cells in the skin. It follows that oral pigmentation is of far more clinical significance in a person of light than in one of dark complexion but is not itself of diagnostic value. Furthermore, many local exogenous pigmentations occur, chiefly from metals, and usually stain the gum margin near the teeth and occasionally the tissues elsewhere in the mouth. Not all mouth pigmentations are due to melanin. Furthermore, melanin pigmentation, if deep in the tissues, may look blue and be confused with cyanosis. Diffuse oral argyria is common. Jaundice is readily detected in the mucous membranes of the mouth, as are all the hemoglobin pigments. Table 4 contains statistics based on the literature that may be of diagnostic value if interpreted in the light of the aforementioned discussion.

TABLE 4. *Conditions Governing Pigmentation of the Oral Mucous Membranes.**

USUALLY PRESENT	OCCASIONALLY PRESENT	RARELY OR NEVER PRESENT	PIGMENTATION OF MOUTH WITHOUT SKIN PIGMENTATION§¶
Pallor (anemia)	Gaucher's disease	Carotenemia	Lead (Burtonian) line
Cyanosis (reduced hemoglobin)	Niemann–Pick's disease	Sprue	Bismuth line‡
Erythremia	Vagabonds' disease	Hodgkin's disease	Argyria
Jaundice	Pernicious anemia†	Chrysiasis	Hydrargyria
Addison's disease	Hemochromatosis	Pellagra	Chalcosis (copper pigmentation)
Argyria	Arsenic poisoning	Porphyria	Zinc pigmentation
Acanthosis nigricans	Scleroderma		Charcoal (in tooth powder)
Lead poisoning	Tuberculosis		Smokers' patch
Bismuthia	Malaria		Tobacco chewing
Normally (darker racial groups)	Cachexia		
Methemoglobinemia	Hyperthyroidism		
Sulfhemoglobinemia			
Carboxyhemoglobinemia			

*The tabulation based on general medical literature. Sufficient data to classify every pigmentary disorder are not available.
†Frequency appears less in the current literature than in the older literature.
‡Bismuth pigmentation of skin (bismuthia) is a great rarity.
§Metals usually present a metallic sulfide line in the gums near the teeth, which is occasionally present more diffusely.
¶For further information on this group see references.[381–384]

External pigmentation of eye. The accompanying presence, and in some cases the persistent absence, of pigmentation of the various external membranes of the eye — the palpebral and bulbar conjunctivas, sclera and cornea — as related to various skin pigmentations is of considerable general clinical interest and often of diagnostic aid.[108, 183, 226, 367, 387, 388] At times the nature of the extraocular pigmentary syndrome is pathognomonic. Examination of the eye must be made in a good light, not omitting an oblique view. The use of

a magnifying lens is helpful. Co-operation with an ophthalmologist to secure inspection with a slit lamp and corneal microscope may enable one to detect pigmentary deposits long before they are evident in the skin, for example, in ochronosis and argyria. Observation with fluorescent light enables pigmentation not visible in ordinary light to be seen.[307] Table 5 contains information that

TABLE 5. *Diagnostic Value of the Association of the Absence or Presence of Pigmentation of the External Portion of the Eye with Skin Pigmentation.**

PIGMENTATION OF EYE AND SKIN	PIGMENTATION OF SKIN WITHOUT PIGMENTATION OF EYE	PIGMENTATION OF EYE WITHOUT PIGMENTATION OF SKIN†
Jaundice	Carotenemia	Blue scleras of osteogenesis imperfecta
Ochronosis	Atabrine pigmentation	Kayser–Fleischer ring of Wilson's disease
Gaucher's disease		Pigmentation of chromium workers
Hemochromatosis (less than 15 per cent)		Chalcosis (copper pigmentation)
Argyria		Siderosis bulbi
Chrysiasis		
Bismuthia		
Melanin pigmentation‡		
Pseudojaundice (picric acid pigmentation)		
Cyanosis§		
Pallor (anemia)		

*Sufficient data to classify every pigmentary disorder are not available; only the combinations found fairly consistently are listed.
†See references [307, 308] for further data on these conditions.
‡Melanin pigmentations of the skin are only occasionally associated with extraocular melanosis.
§Cyanosis is more difficult to detect in the conjunctivas than in the mucous membranes of the mouth.

may be of some aid in orienting the reader to the diagnostic possibilities of the associations mentioned. The lists are by no means complete, since clear-cut statements concerning extraocular pigmentation in many pigmentary disorders are not given in the available literature. It should not be overlooked that melanoblasts have been demonstrated in the conjunctival mucosa[108] and that in certain diseases they are potentially capable of causing a clinically detectable melanin pigmentation. It is not clear from the literature how frequently extraocular melanosis occurs in the various diseases with skin melanosis. It should not be confused with the pigmentary disturbances of the retina and choroid, for example, retinitis pigmentosa. Rarely a systemic disorder such as Addison's disease causes pigmentation of the retina that can be seen on ophthalmoscopic examination.[389]

Pigmentation of fingernails. In contrast to the observation of the mouth and eyes, that for changes in the color of the fingernails in systemic disorders with pigmentation is of limited clinical value. That the fingernails may be pigmented was well shown by Monash,[390] who found that such pigmentation in Negroes is a normal finding of common occurrence, usually in the form of pigmented longitudinal stripes, and in darker-skinned Negroes in that of a diffuse pigmentation in addition. Pigmentation of the fingernails may occur in white persons with disorders likely to produce marked melanosis of the skin, such as Addison's disease[391] (see illustration[246]). Mees's stripe in arsenic poisoning[213, 392] may be simulated by other white striae[393, 394] (see colored plate[394]). The striped area when shed can be analyzed for arsenic. Argyria causes pigmentation resembling cyanosis, but capillaroscopic examination of the nailbeds reveals a normal capillary blood flow.[395] Gold therapy is said to pigment the nails.[391] Pigmentation of the fingernails during hyperthyroidism has been reported.[396] The use of phenolphthalein is listed as rarely causing a bluish discoloration of the fingernails.[397]

LABORATORY DIAGNOSIS OF THE CAUSES OF SKIN PIGMENTATION

The limitations of the human eye in analyzing the causes of skin color changes should be apparent from what has been written. Spectrophotometric analysis of the skin allows recognition of both the amount and nature of the pigments present, but unfortunately the complicated technic and costly apparatus required limit its use to research studies. Under fluorescent light, some pigmentations not visible to the eye in daylight or artificial light can be readily seen (see illustration[307]). A routine history and physical examination and the ordinary laboratory work clarify the cause in most cases. The red-cell count and hemoglobin determination reveal an increase or diminution of hemoglobin in the blood. Spectroscopic examination of the blood detects methhemoglobinocythemia, sulfhemoglobinocythemia, carboxyhemoglobinemia and methemalbuminemia. Cyanosis not due to other hemoglobin derivatives is caused by increased amounts of reduced hemoglobin. Porphyrins can be detected in the urine in porphyria by spectroscopic examination. The quantitative van den Bergh test of serum for jaundice and the Greene-Blackford test for carotenemia are highly specific. Picramic acid and atabrine can be detected chemically in the urine. Rous's test of the urine for hemochromatosis, although not entirely specific, is frequently employed. All these tests are for pigments present in the blood or urine. The Brugsch test[42] and the histamine wheal test for jaundice, as well as the Fishback test[340] for hemochromatosis, are skin tests occasionally used.

Pigments deposited in the skin tissues may be studied by histologic examination of tissue obtained by biopsy. In a general hospital this method is rarely utilized to its full advantage. Requests are usually made by the physician, who understands but little of dermatology and less of the methods and limitation of histologic study of the skin. Skin biopsy specimens are often taken by a surgeon who not only has a limited knowledge of skin pigmentation but is often further handicapped by being given no specific directions concerning the most desirable

place from which to take the specimen. Unless studies are specifically requested, the pathology department is likely to fix and stain these specimens in the same manner as other types of biopsy material.

It is generally agreed by dermopathologists that skin biopsy specimens routinely fixed in formaldehyde and stained with hematoxylin and eosin are not adequate for the study of cytology and especially for evaluation of the cause of pigmentation.[398] Bloch's Dopa reaction to detect activity of melanoblasts,[16] Masson's trichrome stain for cellular detail,[399] silver stains to demonstrate localization and extent of melanin pigmentation, Osborne's method for demonstrating arsenic crystals in the skin,[217] special fixatives for skin tissue, dark-field examination of sections lightly stained with polychrome methylene blue, which enables one to visualize the various metallic pigment granules, as in argyria and chrysiasis, as refractile granules and excludes the nonrefractile granules of melanin and hemosiderin, spectrographic examination of biopsy tissue for detection of metallic pigments,[350] Mallory's potassium ferrocyanide method for staining hemosiderin and Mallory's fuschin stain for hemofuscin are typical of the many technical approaches available to the skilled dermopathologist. It is apparent from this review that the cause of many types of melanin pigmentation cannot be determined histologically and that diagnosis depends chiefly on routine clinical and indicated laboratory procedures.

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MASSACHUSETTS MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND SIXTY-THIRD ANNIVERSARY

May 22, 23 and 24, 1944

THE one hundred and sixty-third anniversary of the Massachusetts Medical Society was observed in Boston on Monday, Tuesday and Wednesday, May 22, 23, and 24, 1944, at the Hotel Statler.

Fourteen hundred and forty physicians were registered.

Monday, May 22

The supervising censors met at 5:00 p.m. in Parlor D.

The Cotting Supper was served to 197 councilors in Parlors A and B. The annual meeting of the Council was held in the Georgian Room, at 7:00 p.m. There were 218 councilors in attendance.

Tuesday, May 23

The first general session was presided over by Dr. George L. Schadt and Dr. G. Lynde Gately, acting as chairman and co-chairman, respectively. The attendance was as high as 650.

This session took the form of a symposium on war injuries.

The one hundred and sixty-third annual meeting of the Society was held at 11:00 a.m. in the Georgian Room. Dr. Roger I. Lee presided. The attendance was 450.

Dr. Lee spoke at length on the state of the Society. The annual oration entitled "A Toxic Factor in Experimental Traumatic Shock," was delivered by Dr. Joseph C. Aub.

During the course of the meeting the President presented Dr. George Mathews, of Providence, delegate from the Rhode Island Medical Society, and Dr. G. Gardiner Russell, of Hartford, delegate from the Connecticut State Medical Society. They responded fittingly.

The annual luncheon was served in Parlors A, B and C to 156 fellows.

A clinical meeting was held in the Georgian Room from 2:00 to 4:00 p.m., under the chairmanship and co-chairmanship of Dr. Dwight O'Hara and Dr. Clifton T. Perkins, respectively. The attendance was 600.

The Shattuck Lecture was delivered at 4:00 p.m. by Dr. Alfred Blalock, of Baltimore, Maryland. Dr. Blalock's subject was "A Consideration of Recent Advances in Surgery." (This lecture will appear in the *New England Journal of Medicine*, issues of August 17 and 24.)

The annual dinner was held in the Georgian Room at 7:00 p.m. with 492 in attendance. This dinner was graced by the presence of the Honorable Leverett Saltonstall, Governor of Massachusetts. William Cunningham, of the *Boston Herald*, was the principal speaker.

Wednesday, May 24

There was a general clinical meeting in the Georgian Room from 9:00 a.m. to noon under the chairmanship and co-chairmanship of Dr. Charles F. Wilinsky and Dr. Leroy E. Parkins, respectively. The attendance was 600.

From 12:00 noon until 2:00 p.m. a series of section meetings and luncheons was held.

The Section of Medicine, under the chairmanship of Dr. George D. Henderson, met in Parlor B. Because of the numbers attending, it was adjourned to the Georgian Room. Sixty-two sat down for luncheon, and approximately 300 attended the clinical meeting afterward.

The Section of Surgery, under the chairmanship of Dr. Howard M. Clute, met in Parlor C. The attendance was 65.

The Section of Pediatrics, under the chairmanship of Dr. Leroy T. Stokes, met at The Junior League, Zero Marlboro Street. The attendance was 54.

The Section of Obstetrics and Gynecology met in Parlor A, under the chairmanship of Dr. Christopher J. Duncan. The attendance was 62.

The Section of Radiology met in Room 716 W, under the chairmanship of Dr. Stanley A. Wilson. The attendance was 25.

The Section of Physiotherapy met in Parlor E, under the chairmanship of Dr. Wilmot L. Marden. The attendance was 13.

The Section of Dermatology and Syphilology met in the Hancock Room, under the chairmanship of Dr. John G. Downing. The attendance was 35.

The New England Society of Anesthesiology met in Parlor D. The attendance was 32.

From 2:00 to 5:00 p.m. a general clinical meeting was held in the Georgian Room under the chairmanship and co-chairmanship of Dr. Daniel B. Reardon and Dr. Vlado A. Getting, respectively. The attendance varied from 450 to 600.

There were eighteen scientific and seventy-six technical exhibits.

The motion pictures, as usual, attracted a large number.

The official list of officers, standing and special committees, councilors, censors, admissions, deaths and so forth are appended.

Michael A. Tighe, *Secretary*

Annual Meeting of the Society

The one hundred and sixty-third annual meeting of the Massachusetts Medical Society was called to order by the president, Dr. Roger I. Lee, in the Georgian Room of the Hotel Statler, Boston, at 11:00 a.m., May 23, 1944. There were approximately 450 fellows present.

The Secretary offered the record of the last annual meeting as published in the *New England Journal of Medicine*, issue of July 22, 1943. There being no objection, this report was declared accepted by the chair.

Dr. Lee presented Dr. George Mathews, of Providence, representative of the Rhode Island Medical Society. Dr. Mathews brought the greetings of his society.

Dr. G. Gardiner Russell, of Hartford, Connecticut, was introduced and brought the greetings of the Connecticut State Medical Society.

The Secretary announced that the membership of the Massachusetts Medical Society, as of May 25, 1943, was 5753, and as of May 23, 1944, 5778. He said that 104 fellows had died, 11 had resigned and 1 had been deprived of membership during this interval. He added that 129 new fellows had been created and that 12 had been reinstated during this period. The net gain in membership, he said, was 25.

The following amendments to the by-laws were read by Dr. Lee:

> Chapter IV, Section 4, of the by-laws of the Massachusetts Medical Society is amended by striking out the word "stated" appearing in the first line and inserting in its place the word "annual," by deleting the words "in October" appearing in the second line, by striking out the word "stated" in next to the last line and inserting in its place the word "annual" and by deleting in the last line the words "in February."
>
> The amended section reads as follows:
>
> Section 4. The Council shall elect at the annual meeting, on nomination by the President or from the floor, the Auditing Committee, composed of two fellows who are not councilors.
>
> This committee following the close of the fiscal year, shall require by a certified public accountant an examination of the assets and securities of the Society in the custody of the Treasurer, and of the Treasurer's books and accounts.
>
> This committee shall verify the accountant's examination and report its findings at the annual meeting of the Council.
>
> Chapter VI, Section 5, of the by-laws of the Massachusetts Medical Society is amended by striking out the word "February," as it appears in the fifth line of page 21, and inserting in its place the word "annual."
>
> This amended paragraph reads as follows:
>
> He [the Treasurer] shall render to the Council at its annual meeting a full written report of the assets and liabilities on December 31 of the previous year, and also of the financial transactions of the Society during that year.

Dr. Lee said that the purpose of these changes was to allow more time for the auditors to do the job assigned them under the by-laws. He added that they had been favorably acted upon by the Council on May 24, 1943.

Dr. Frank R. Ober, Suffolk, moved the adoption of these two amendments. This motion was seconded by Dr. Clarence E. Burt, Bristol South, and it was so ordered by vote of the members.

Dr. Lee called on Dr. William H. Robey, Suffolk. He presented resolutions on the death of Dr. Charles S. Butler, former treasurer of the Massachusetts Medical Society. These resolutions appear on

the records of the meeting of the Council held on May 22, 1944.

Dr. Lee made an urgent plea to the district secretaries to send to Dr. Tighe the names of all physicians who had entered the armed services from their respective districts.

Dr. Lee called on the vice-president, Dr. Daniel B. Reardon, Norfolk South, to assume the chair. In so doing, Dr. Reardon spoke as follows:

As your vice-president, it has been my privilege and pleasure for the past year to work with our president. I have found him an untiring, zealous and hard-working individual. He has not alone confined his time and efforts to the welfare of the Massachusetts Medical Society, but as you well know, he is chairman of the Board of Trustees of the American Medical Association. To both these offices he has given his time and efforts for the welfare of the medical profession and medicine in general.

It is now my distinct honor and pleasure to give you Dr. Roger I. Lee, president of the Massachusetts Medical Society, who will report on the state of the Society.

Dr. Lee responded as follows:

It is a happy custom that the president of the Massachusetts Medical Society reports on the affairs of the Society at the annual meeting.

Your present incumbent finds great pleasure in making his report. Any report at this time, however, is made under the shadow of a grim catastrophe that has enveloped the entire world. It has dislocated all our lives. It has dominated all our thoughts. Of necessity, winning the war is the sole objective of all of us. We that are here are under orders if in uniform, under sentence if in civilian clothes. Perhaps a third of the fellows of this society are in uniform, and before long, as the war continues and as recent graduates go into the armed services, approximately half the active physicians of the Commonwealth will be in uniform. It is, I believe, an extraordinary tribute to the unwavering loyalty and the altruistic idealism of the medical profession that no attempt has been made to capitalize on the pressing necessity for doctors in the armed forces or on the resulting shortage of doctors on the home front. There are no doctors' blocs seeking special class legislation. There are no doctors' strikes. There are no doctor profiteers. This is no doctor "black market." And I desire to pay tribute to the wives and families of the doctors in the service. Doctors volunteer. There has been no drafting of doctors, unless one means that a man's conscience drafted him. Doctors volunteered for their soldiers' pay, not for "cost plus" and not for war workers' pay, and their wives make what shift they can. And when these men come back, let these sacrifices be not forgotten.

And in passing may I give a bit of tribute to the wives of the doctors who stayed at home! They have done double duty, quite unheralded and quite unsung. And most of the doctors' patients have, in this trying situation, been understanding and co-operative, and have cheerfully foregone on many occasions the comfort of the doctor's visit, if in his judgment a visit was not absolutely necessary.

The Massachusetts Medical Society operates through committees, which report to the Council. Four of these committees are composed of one man from each district society, elected by the district society. These are the Nominating Committee, although technically not a committee, the Committee on Public Relations, the Executive Committee and the Committee on Legislation. The nominating councilors from each district have the sole function of nominating the officers of the Society. The Committee on Public Relations is perhaps a dozen years old. This committee has been so strikingly useful along the lines of its designation that two quite new committees — the Executive Committee and the Committee on Legislation — have been set up along similar but not identical lines for their particular purposes. The president of the Society, who is an *ex-officio* member of all committees, is named as the chairman of the Committee on Public Relations and of the Executive Committee. It is not my purpose to repeat the story of the activities of the various committees; it is rather to discuss the functioning of the committee mechanism of the Society, inasmuch as the Society operates through committees reporting to the Council.

The Executive Committee was designed for two purposes: to free the Council from considerable detail and to provide a group, informed before the Council meeting, concerning certain committee reports and other items of a complex nature. Although there has been some confusion about the extent of the authority of the Executive Committee, it is my considered opinion that this committee, which has worked very diligently, has contributed much to the thorough consideration of problems so that the Council might act wisely. It is, I believe, of great benefit to the Society that free preliminary discussion of certain questions can take place in a large representative committee like the Executive Committee, and indeed, certain problems are gone over by the Committee on Public Relations before consideration by the Executive Committee.

These four committees, excluding the ex-officio members, are composed of seventy-two men. Naturally some of these are duplicates. On the other hand, there are alternate members of the Executive Committee. And this number refers only to four committees, all elected by the member districts.

Most of the committees of the Society, whether standing or temporary, are small. The ordinary procedure is that the president nominates and the Council elects. The Council may also nominate. Most committees consist of five members. But there are larger committees, notably a committee of seventeen, appointed to consider a loan fund for returning medical officers of the Society. There are around 250 fellows serving on all these committees. The majority of the members of the committees are members of the Council. The chairmen of the standing committees are *ipso facto* members of the Council. The Council numbers at this time 328. The number of fellows in the Society is about 5778.

Your president has always believed that service on the committees of the Society was both an obligation and an opportunity and should be freely shared by the fellows and that committee membership should be frequently changed. He also has strongly believed in the liberal mixture of young and old in committees. Alas for his theories. The war took off the younger men. Older men, familiar with certain committee duties, could carry on effectively with a minimum of effort. So, very regretfully, committees were frozen. But when the war is over and the fellows come back, I want this to be a public record of what one president feels regarding committee changes. I should like also to suggest a slower pace of change in some of the committees, such as the Committee on Ethics and Discipline and the Committee on Finance. I believe, too, that this should also apply to the delegates to the House of Delegates of the American Medical Association. I do not believe, however, that any committee should be regarded or regard itself as fixed.

Each of the Society's committees has made a report to the Council. These reports will be printed in the *Journal* in the near future and therefore will not be considered here.

Reports may not be very revealing. The Committee on Ethics and Discipline has a talent that approaches genius in laconic statement. Its activities are mostly confidential. The time spent by this committee is enormous. The good to the Society is incalculable.

A recent committee that has labored quietly but effectively is the Medical Advisory Committee to the Regional OPA. No fire alarms, no sirens, but some hefty spade work and a lot of sweat, and a good crop at the end. Washington says it is the best advisory committee in the country.

During this session, we have had frequent cause to be grateful to the Committee on Arrangements. Dr. Gordon Morrison reluctantly consented to remain on the job again this year. He and his hard-working committee have done a grand job. What does not appear on the surface is the liaison that Dr. Morrison has maintained between his

committee and the committees of other organizations on war meetings.

I saw Dr. Walter P. Bowers the other evening in Worcester. He said to me, "Now, don't you ever forget that I picked out Nye as editor of the *Journal*." I had thought that the Committee on Publications had picked Nye. Bill Breed claims that he discovered Nye. Under the ownership of the Massachusetts Medical Society, the *Journal* (by which I mean, of course, the *New England Journal of Medicine*) has had but two editors — Dr. Bowers and Dr. Nye. The Committee on Publications selects the Shattuck Lecturer and gives some heed to the publication of the *Directory*, and ostensibly to the *Journal*. I have been a member of that committee with Dr. Bowers as editor and with Dr. Nye as editor. I seem to recall that I announced to the Council the appointment of Dr. Nye. But, in any event, the *Journal* has prospered wonderfully in these war times. As you know, fellows in the armed forces are excused from dues, including subscription to the *Journal*. Some of us thought that, with diminished subscriptions, paper shortage and inability to take advertising, the Society might be in for a considerable loss in the *Journal* account. But, at the moment, the circulation is increasing. As a humble contributor, I can testify that requests for reprints come from nearly every state in the Union. You have every reason to be proud of the *Journal*, the editor, the editor emeritus and the Committee on Publications.

The financial status of the Society seems excellent. The new treasurer, Dr. Hubbard, has given generously of his time and energy. There is now set up a system of expert advice on securities and of modern accounting.

The additional space for the officers of the Society has permitted a considerably increased volume of work of the Society to be carried on smoothly. For example, there are sometimes as many as four committees of the Society meeting at once. Wednesday seems to be the day that the doctor takes a busman's holiday and goes to 8 Fenway, Boston, for a committee meeting of the Massachusetts Medical Society.

As you may know, plans are already under active discussion to establish an information bureau at the Society's headquarters.

As a part of his duties, the president has the privilege of visiting the district societies. This was indeed a pleasure, and it is a matter of deep regret that it was not possible to visit all the district societies. Despite the handicaps and obstacles of wartime conditions, the district societies have been able to continue to hold interesting meetings with good attendance.

Under the authority of the Executive Committee, which was subsequently approved by the Council, a committee from the Society met several times with representatives of the other New England State medical societies for discussion of legislative matters. This idea did not originate in this state. It seems to be generally true, if the New England states are a criterion, that doctors are largely uninformed concerning possible legislation involving the practice of medicine. Being uninformed the profession is often unable to bring to any discussion the benefits of its experience. Then, too, the uninformed are frequently led into unwise, irrelevant and perhaps inaccurate remarks, which are not helpful to orderly discussion. It would seem the plain duty of every member of the profession to acquaint himself fully with the various and sundry legislative proposals; he should study these proposals in the light of his own knowledge of the facts. Some of these problems are today as unanswerable as the cure of cancer or the prevention of the common cold.

Doctors are citizens and as such are entitled to political beliefs and political affiliations. But in the discussion of medical service to the people, political beliefs and political affiliation have no place. So long as the Massachusetts Medical Society is recognized as unselfish and altruistic, just so long can it expect to have its voice listened to with respect.

It is pleasant to record improvement in the relations between the Society and the regularly instituted legislative, executive and health authorities on Beacon Hill. The medical profession has no selfish interest of its own to further. It does have a wealth of experience and of knowledge that should be made available for the benefit of the public. The past year promises to be epochal in the standards of medical practice in Massachusetts, and if nothing unforeseen happens, the year 1944 will deliver the Commonwealth of Massachusetts from its unenviable position of being the dumping ground of graduates, actual, nominal or what you will, of unapproved medical schools. There is now an excellent Board of Registration in Medicine. All this comes as the result of years of effort to secure a good medical-practice Act, which is now the law in Massachusetts. In this state, medical standards have suffered grievously because of excessive lay dominance over the medical profession. In a recent address at the one hundred and fiftieth anniversary of the Worcester District Medical Society, I took occasion to point out how lay thought and lay influence had controlled the medical profession from the days of the medicine man to the present day. The best medical school in the country, the best hospital in the country or the best and most highbrow medical society in the country does not set medical standards; but legislatures do. I am far from advocating unrestricted medical control. I admit that penicillin is a product of war conditions and that it is doubtful how far penicillin would have been developed without the war. One seemingly cannot have penicillin without the war. But my sole point is that doctors do not have sufficient voice in the determination of many medical problems, particularly that concerning the standards of medical practice.

There are many fellows of the Society who have made the past year a pleasant one for the outgoing president. There is one person, not a fellow, to whom I have been indebted on many occasions for never-failing courteous co-operation, our executive secretary, Bob Boyd. And there is one fellow, who is in one person a seeing eye, a watchdog, a life-saving St. Bernard, and man's and the president's best friend — the secretary of the Society, Mike Tighe.

(Dr. Lee's address, which showed not only an intimate knowledge of the Society in all its affairs but likewise a very human understanding of the problems with which the war has confronted its members, was greeted with loud applause.)

Dr. Lee resumed the chair and introduced the new officers:

President: Elmer S. Bagnall, Essex North
Vice-president: Sumner H. Remick, Middlesex South
President-elect: Reginald Fitz, Suffolk
Secretary: Michael A. Tighe, Middlesex North
Treasurer: Eliot Hubbard, Jr., Middlesex South
Assistant treasurer: Norman A. Welch, Norfolk
Orator: Frank H. Lahey, Suffolk

All responded fittingly.

The President expressed his pleasure in presenting the distinguished orator, Dr. Joseph C. Aub, of Boston, whose subject was "A Toxic Factor in Experimental Traumatic Shock." (At the close of his oration Dr. Aub was loudly applauded. His oration will appear in the *New England Journal of Medicine*, issue of July 20, 1944.)

Dr. Reardon moved that the meeting adjourn. This motion was seconded by a member, and it was so voted.

Dr. Lee declared the one hundred and sixty-third annual meeting of the Massachusetts Medical Society adjourned at 12:20 p.m.

MICHAEL A. TIGHE, *Secretary*

APPENDIX

Admissions Recorded from May 25, 1943, to May 23, 1944

YEAR OF ADMISSION	NAME AND RESIDENCE	MEDICAL SCHOOL
1944	Alba, Anthony, Dorchester	Tufts
1943	Alpert, David Robert, Allston	Tufts
1943	*Alifano, John James, Springfield	Middlesex
1943	Anderson, Donald Grigg, Boston	Columbia University
1943	Anderson, Dorothy Sperling, Newton Lower Falls	Boston University
1944	Anderson, Erna Goettsch, Boston	Columbia University
1943	*Annis, Israel Alan, Auburndale	Middlesex
1943	Armstrong, Sinclair Howard, Jr., Boston	Harvard
1944	*Avallone, Louis John, Somerville	Middlesex
1943	Baker, Joseph Michael, Springfield	University of Vermont
1944	Bandeian, John Jacob, Bridgewater	Tufts
1943	Barry, Herbert, Jr., Cambridge	Tufts
1944	Beals, Lester H., Mount Hermon	University of Michigan
1944	Belin, Harry, Middleton	Tufts
1943	*Berman, Leo, Brookline	Basel University
1944	Blackmer, Lewis Dwight, Quincy	Tufts
1944	Blodgett, James Bishop, Brookline	Harvard
1943	Boone, Edward William, Boston	Harvard
1943	Booth, Marion Frances, Northampton	Univerity of Nebraska
1943	Boynton, Willard Harold, East Boston	Tufts
1944	Bruno, Francis Ernest, Quincy	Tufts
1943	Bryan, Charles Silas, Jr., Cambridge	Harvard
1943	*Butts, Vincent, East Boston	College of Physicians and Surgeons, Boston
1944	Cahill, George Byron, Boston	University of Minnesota
1944	Carroll, Robert Ernst, Boston	Yale
1944	Chapman, William Phalen, Boston	McGill
1943	*Chase, Frank Hills, Stoneham	Bellevue Hospital Medical College
1944	*Chesanow, Albert, Great Barrington	Middlesex
1944	*Ciampa, Frank, Somerville	University of Naples
1943	*Civen, Eva, Dorchester	Middlesex
1944	Congdon, Frederick Richard, Pittsfield	Tufts
1943	Davidson, George Davis, Quincy	Boston University
1944	*Davis, Robert Armstrong, Wareham	Mid-West Medical College
1943	Dopkeen, Saul Kahn	Boston University
1944	*Drury, George Eliot, Malden	College of Physicians and Surgeons, Boston
1944	*Ehrentheil, Otto Felix, Brighton	University of Vienna
1943	*Ehrlich, Robert, Newton Centre	Middlesex
1943	Engleman, Clarence Clarke, Brookline	Western Reserve
1943	Ewing, Edward Hilts, Stoughton	College of Physicians and Surgeons, Baltimore
1943	Fell, James Edward, Fall River	Tufts
1944	Finch, Clement Alfred, Boston	University of Rochester
1943	Floyd, Louis Carl, Brookline	University of Texas
1943	Flynn, James Lawrence, Boston	Georgetown
1943	Flynn, John F., Pittsfield	Washington University
1943	*Friedman, Sidney, Brookline	Middlesex
1943	Garrett, John William Douglas, Boston	University of Toronto
1943	Goeringer, Claire Ferdinand, Fort Devens	Johns Hopkins
1943	Hall, Albert Eden, Worcester	Long Island
1944	Halter, Paul Edmund, Boston	Western Reserve
1944	Heerdegen, Dorothy Kathryn, Jamaica Plain	Ohio State University
1944	Heimlich, Fred, Brookline	Tufts
1943	Hemenway, Ruth Victoria, Williamsburg	Tufts
1943	*Hite, Jacob William, Boston	Middlesex
1943	*Hochman, George Victor, Sharon	Middlesex
1944	Holt, William Leland, Amherst	Harvard
1943	Howard, Rutledge William, Boston	Harvard
1944	Howe, Chester W., Brighton	Tufts
1943	Hudson, Henry Alfred, Marblehead	Tufts
1943	*Johnson, Daniel Philip, Melrose	University of Lausanne
1943	Kasdon, Solomon Charles, Boston	Yale
1944	Katz, Harvey Warren, Everett	Tufts
1943	*Kay, Stephen Benjamin, Fitchburg	Middlesex
1944	Kenney, Francis R., West Newton	Boston University
1944	Landis, Eugene Markley, Chestnut Hill	University of Pennsylvania
1944	Leighton, Herbert Towle, Brookline	Harvard
1943	Le Vine, Bernard Elliot, Roxbury	Tufts
1944	Levine, Irving Maxwell, Dorchester	Tufts
1944	*Levy, David Abraham, Mattapan	Middlesex
1943	Lincoln, James Rufus, Wareham	Harvard
1944	Lockhart, Arthur John, Chestnut Hill	Columbia University
1944	Lodge, Edmund Anderson, Gloucester	University of Kansas
1943	Lynam, Patrick Joseph, Malden	Tufts
1943	*Magendantz, Heinz Herbert, Newton Highlands	University of Cologne
1944	*Makofski, Anthony B., Northampton	Kansas City University of Physicians and Surgeons
1944	*Marks, Abraham S., Malden	Middlesex

1943	McCarty, William Charles, Cambridge	Harvard
1943	McGillicuddy, John Joseph, Brighton	Yale
1944	McLaughlin, James Alphonsus, Ocean Bluff	Tufts
1943	McNulty, John Joseph, Roslindale	Tufts
1943	Meissner, William Avison, Brookline	University of Oregon
1944	Melcher, Margaret S., Northampton	Stanford
1943	*Michalski, Kasimir Stanislaw, Worcester	Middlesex
1943	Miller, Daniel, Boston	Tufts
1943	Miller, George Francis, Dorchester	Harvard
1943	*Minsky, Joseph William, West Boylston	Mid-West Medical College
1943	Money, Isabel Stirling, Mattapan	Boston University
1944	Moore, Francis Daniels, Brookline	Harvard
1944	Moss, Robert Elmer, Boston	Boston University
1943	Murphy, Albert Summers, Waban	Harvard
1943	Murphy, John Anthony, Boston	Tufts
1944	Murray, Jean, Boston	Johns Hopkins
1944	Myers, Robert Samuel, Brookline	Harvard
1944	*Neustadt, Else, Westwood	University of Leipzig
1944	*Neustadt, Rudolph, Westwood	University of Leipzig
1943	Nevin, Robert Williston, Vineyard Haven	University of Vermont
1944	O'Keeffe, James Vincent, Lynn	Harvard
1944	Ostrov, Norman N., Dorchester	Tufts
1943	Parker, Arthur Seymour, Jr., Brookline	Georgetown
1943	Parrish, Carroll Ernest, Melrose	College of Medical Evangelists
1944	Porter, Charles Talbot, Pepperell	Harvard
1943	*Portner, Irwin Isaac, Brighton	Middlesex
1943	Prince, Anna Agassiz, South Hamilton	Columbia University
1944	Provenzano, R. William, Jamaica Plain	Tufts
1944	Quinby, John Thayer, Brookline	Harvard
1943	Rice, John Dexter, Shrewsbury	Tufts
1944	Robinson, Hugh Laughlin, West Newton	Harvard
1943	Rogers, Lois Knight, Concord	Yale
1944	Rooney, John James, Waban	Columbia University
1944	*Rosenthal, Myer, Mattapan	Middlesex
1944	Saunders, Allen Irving, Roxbury	Tufts
1943	Shapiro, Robert Rubin, Brookline	Harvard
1944	*Shepard, Herbert Norman, Allston	Middlesex
1944	Sheridan, Edward Paul, Boston	Northwestern University
1944	Silverstein, Maurice L., Dorchester	Boston University
1943	Stearns, Samuel, Dorchester	University of Michigan
1944	Stratton, Harold Linson, Worcester	Tufts
1944	Taylor, Lois Ellene, Belchertown	University of Vermont
1944	Thomson, Robert Strachan, Walpole	Harvard
1944	Trafton, Howard Munro, Hyde Park	Boston University
1944	Tucker, Arthur Wallace, Jr., Needham	Harvard
1944	Tyson, Dudley Ball, Boston	Tufts
1944	*Valicenti, Peter Roger, Rockland	Middlesex
1943	Van Dyke, Wilhelmina Marie, East Milton	University of Michigan
1943	Varnerin, Emma Mary, Roxbury	Boston University
1943	*Vilker, Arthur Hyman, Revere	Middlesex
1944	Votta, Ipoldi John, Boston	Boston University
1943	Waldman, J. Edward, Malden	Tufts
1943	*Werby, Isidore, Brighton	Middlesex
1944	Zonderman, Bernard, Revere	Boston University

Total 129

*The candidate after a personal interview, was approved by the Committee on Membership and permitted to take an examination before a board of censors.

Deaths Reported from May 23, 1943, to May 23, 1944

ADMITTED	NAME	PLACE OF DEATH	DATE OF DEATH	AGE
1910	†Abbe, Elizabeth Morrison	Roxbury	January 18, 1943	68
1900	†Alfred, James	Brockton	June 12, 1943	69
1883	†Atwood, Charles Augustus	Taunton	October 10, 1943	82
1939	Barrett, James Aloysius	New Bedford	April 28, 1944	58
1941	Barry, Thomas A.	"In the service of his country"	August 4, 1943	34
1914	Bell, Clarence John	Wellfleet	June 6, 1943	67
1890	†Blake, John Bapst	Brattleboro, Vermont	August 17, 1943	77
1912	Brady, William Francis	St. Petersburg, Florida	February 28, 1944	58
1887	†Brennan, John Joseph	Worcester	August 26, 1943	79
1908	Broderick, Francis Patrick	Jamaica Plain	February 26, 1943	67
1899	Buehler, George Van Buskirk	Bedford	November 19, 1943	67
1921	Campbell, Charles Macfie	Cambridge	August 7, 1943	66
1898	Carden, Charles James	Tewksbury	July 4, 1943	70
1934	Chase, Lawrence Milton	Carver	February 8, 1944	60
1915	Clarke, Willis Earl	West Somerville	February 23, 1944	56

1925	Cochran, John Joseph	Natick	May 1, 1944	47
1921	†Colburn, Frederick Wilkinson	Boston	April 9, 1944	73
1925	Connor, Thomas John	Arlington	July 16, 1943	51
1907	Coriat, Isador Henry	Boston	May 26, 1943	67
1895	Coues, William Pearce	Brookline	January 13, 1944	71
1927	Cronin, Edward Joseph	Brighton	February 11, 1944	46
1919	Curran, Louis Frederic	Dorchester	July 22, 1943	57
1911	Cushman, Howard Lewis	Methuen	November 3, 1943	57
1907	Dascombe, Otho Lee	Waltham	September 26, 1943	63
1900	†Daudelin, Simeon Alphonse	Montreal, Canada	August 28, 1943	73
1936	David, Ernest Joseph	Lowell	July 11, 1943	59
1903	Donaldson, James Frank	Middleton	March 10, 1944	66
1936	Dore, Rev. Francis James (Honorary Fellow)	Chestnut Hill	February 28, 1944	67
1897	Egan, John Joseph	Gloucester	January 22, 1944	73
1903	Elliott, Alfred	Middleboro	March 1, 1943	71
1893	†Emerson, Francis Patten	Franklin	January 19, 1944	81
1938	Faillace, Gaetano	Brookline	February 19, 1944	71
1938	Flanagan, Lt. James Edward (MC), U.S.N.R.	"Reported missing in action"	January 2, 1944	31
1884	Foster, Charles Chauncy	Cambridge	December 2, 1943	85
1911	Frank, Morris	Roxbury	June 17, 1943	56
1884	†Gage, James Arthur	Lowell	November 29, 1943	85
1908 1927 1896 1913 1941	Geary, Cornelius Edward	Fitchburg	December 6, 1943	63
	Gerstein, Maurice	Brookline	July 13, 1943	73
1919	Gillespie, Norman Wilkinson	Dorchester	April 28, 1944	55
1924	Givan, Lt. Comdr. James Alexander (MC), U.S.N.R.	Norfolk, Virginia	November 14, 1943	43
1934	†Gleason, Edward Francis	Hyannis	April 9, 1944	75
1933	Graves, Lt. Comdr. Sidney Chase (MC), U.S.N.R.	"Somewhere in the Pacific"	Unknown	43
1910	Hallisey, Joseph Edward	Boston	November 18, 1943	61
1923	Halloran, Col. Roy Dennis, M.C., A.U.S.	Washington, D. C.	November 10, 1943	49
1900	Hart, Henry Brown	Sarasota, Florida	July 13, 1943	72
1906	Hartman, Gustave	Lynn	August 1, 1943	66
1902	Hartwell, Harry Fairbanks	Weston	December 7, 1943	70
1907	Hatch, Ralph Augustus	Brookline	April 1, 1944	62
1885	Howard, Margaret Emily Pagelsen	Reading	March 1, 1944	84
1904	Howe, Harry Newell	Greenfield	May 11, 1944	72
1897	†Jackson, Alton Atwell	Everett	July 25, 1943	89
1911	Jacques, J. Hector	Fitchburg	April 15, 1944	68
1905	Kennedy, Edward Anthony	Pittsfield	December 14, 1943	63
1909	Kennison, Fred Marshman	Boston	July 31, 1943	80
1905	Kent, Ralph Porter	Oak Bluffs	August 5, 1943	63
1898	Kite, Walter Chester	Milton	February 5, 1944	79
1899	†Laighton, Florence Marion	New York City	January 15, 1943	72
1920	Laserte, Charles John	Leominster	April 14, 1944	62
1912	Leland, George Adams, Jr.	Brookline	September 22, 1943	57
1903	Lemaire, Willard Wallace	Worcester	December 27, 1943	64
1937	Lodge, Athens Vallette	Brewster	November 18, 1943	71
1940	Lowenthal, Karl	Fall River	January 16, 1944	51
1902	Lowney, John Francis	Fall River	February 19, 1944	74
1908	Mannix, Louis Edward	Springfield	March 11, 1944	59
1914	Mason, Broadstreet Henry	West Hartford, Connecticut	January 1, 1942	60
1902	Mayhew, Orland Smith	Vineyard Haven	February 26, 1944	66
1921	McAuslan, James Lewis	North Grafton	March 22, 1944	69
1936	Merchant, Raymond Francis	Vineyard Haven	June 7, 1943	41
1936	Merrick, Frank Humphrey	Dorchester	January 17, 1944	37
1928	Miller, Howard Stephen	Taunton	September 6, 1943	55
1911 1939	O'Brien, John Francis	Fall River	June 11, 1943	57
1928	Palmer, Anna Chipman	Brookline	February 10, 1944	86
1887	†Parks, Silas Henry	Great Barrington	November 7, 1943	82
1927	Parlow, George Gibson	Wareham	April 20, 1944	74
1911	Penny, Mary McDermott	Saugus	April 17, 1944	72
1914	Percy, Karlton Goodsell	Brookline	November 15, 1943	57
1898	Perley, Roscoe Damon	Melrose	January 21, 1944	79
1932	Phillips, Major Robert Titus, M.C., A.U.S.	Philippines	Unknown	41
1904	Pote, Leonard Holden	Somerville	December 3, 1943	69
1889	†Pothier, Joseph Charles	New Bedford	December 1, 1943	78
1923	Rich, Herbert Lowell	Attleboro	January 23, 1944	78
1908	Richardson, Edward Peirson	Brookline	January 26, 1944	62
1898	†Richmond, Simon	Roxbury	February 5, 1943	70
1907	Riley, Charles Allen	Chestnut Hill	January 30, 1944	62
1929	Seed, Raymond Charles	Lawrence	June 6, 1943	45
1920	Senecal, Raymond Ernest	New Bedford	August 24, 1943	51
1921 1943	Silberg, Morris Abraham	Roxbury	November 20, 1943	49
1897	†Spalding, Fred Maurice	Brookline	January 24, 1944	73
1919	Spellissy, Frank Thomas	Marlboro	February 4, 1944	48
1925	Stein, Albert	Thompsonville, Connecticut	December 5, 1942	49
1896	Stetson, Halbert Greenleaf	Greenfield	September 15, 1943	75
1920	Stickney, Robert Cole	Beverly	September 7, 1943	50

1905	Stone, Ralph Edgarton	Beverly	March 22, 1944	67
1907	†Sullivan, John Thomas, Jr.	Dorchester	December 10, 1943	70
1925	†Thompson, Clara Louise	Boston	September 16, 1943	72
1882	†Tilton, Josiah Odin	Lexington	December 2, 1943	90
1914	Varney, Elton Murray	Peabody	March 11, 1944	66
1899	Washburn, Frank Hall	Holden	November 10,1943	72
1909	Wentworth, Mark Hunking	Concord	May 15, 1944	65
1923	Winslow, George Edgar	Hyde Park	April 17, 1944	67
1907	Wood, Benjamin Ezra	Canton	December 26, 1943	63
1928	Wright, Allen Henry	East Northfield	June 17, 1943	65
1895	†Wylie, Eugene Cushman	Dorchester	September 15, 1943	71
1915	†Young, Annie Roberts	Waltham	March 16, 1943	79
1924	Yudin, Hyman	Beverly	December 18, 1943	54

†Retired fellow.

Total number of deaths of active fellows	83
Total number of deaths of retired fellows	21
Total number of deaths of honorary fellows	1
Grand total	105

Officers for 1944–1945

President: Elmer S. Bagnall, Groveland, 281 Main Street.

President-Elect: Reginald Fitz, Brookline. Office, Boston, 319 Longwood Avenue.

Vice-President: Sumner H. Remick, Waltham, 735 Trapelo Road.

Secretary: Michael A. Tighe, Lowell. Office, Boston, 8 Fenway.

Treasurer: Eliot Hubbard, Jr., Cambridge, 29 Highland Street.

Assistant Treasurer: Norman A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Avenue.

Orator: Frank H. Lahey, Boston, 605 Commonwealth Avenue.

Committees Elected by the Districts

Executive Committee of the Council — Established 1941 (members *ex-officiis* and one councilor and alternate elected by the councilors of each district medical society).

President: Elmer S. Bagnall, Groveland, 281 Main Street.

President-Elect: Reginald Fitz, Brookline. Office, Boston, 319 Longwood Avenue.

Vice-President: Sumner H. Remick, Waltham, 735 Trapelo Road.

Secretary: Michael A. Tighe, Lowell. Office, Boston, 8 Fenway.

Treasurer: Eliot Hubbard, Jr., Cambridge, 29 Highland Street.

Term Expires 1945

Essex South: Loring Grimes, Swampscott, 84 Humphrey Street. (Alternate: Charles L. Curtis, Salem, 10 Federal Street.)

Hampshire: L. Beverly Pond, Easthampton, 115 Main Street. (Alternate: Joseph D. Collins, Northampton, 187 Main Street.)

Middlesex South: Dwight O'Hara, Waltham. Office, Boston, 416 Huntington Avenue. (Alternate: Arthur M. Jackson, Everett, 512 Broadway.)

Norfolk South: Henry A. Robinson, Hingham, 205 North Street. (Alternate: Nahum R. Pillsbury, South Braintree, Norfolk County Hospital.)

Suffolk: Donald Munro, Boston, 818 Harrison Avenue. (Alternate: Charles C. Lund, Boston, 319 Longwood Avenue.)

Worcester: Ralph S. Perkins, Worcester, 10 Hackfeld Road. (Alternate: Gordon Berry, Worcester, 36 Pleasant Street.)

Term Expires 1946

Barnstable: William D. Kinney, Osterville.

Bristol North: William H. Allen, Mansfield, 70 North Main Street. (Alternate: Ralph M. Chambers, Taunton, Taunton State Hospital.)

Bristol South: Edwin D. Gardner, New Bedford, 150 Cottage Street. (Alternate: Richard B. Butler, Fall River, 278 North Main Street.)

Essex North: Frank W. Snow, Newburyport, 24 Essex Street. (Alternate: Rolf C. Norris, Methuen, 247 Broadway.)

Middlesex East: Edward M. Halligan, Reading, 37 Salem Street. (Alternate: Richard Dutton, Wakefield, 33 Avon Street.)

Plymouth: Peirce H. Leavitt, Brockton, 129 West Elm Street. (Alternate: Charles D. McCann, Brockton, 12 Cottage Street.)

Term Expires 1947

Berkshire: Isaac S. F. Dodd, Pittsfield, 34 Fenn Street. (Alternate: Patrick J. Sullivan, Dalton, 471 Main Sreet.)

Franklin: William J. Pelletier, Turners Falls, 113 Avenue A. (Alternate: Merritt B. Low, Deerfield, 31 Federal Street.)

Hampden: William A. R. Chapin, Springfield, 121 Chestnut Street. (Alternate: Edward P. Bagg, Holyoke, 207 Elm Street.)

Middlesex North: William F. Ryan, Lowell, 219 Central Street. (Alternate: William M. Collins, Lowell, 174 Central Street.)

Norfolk: Charles J. Kickham, Brookline. Office, Boston, 524 Commonwealth Avenue. (Alternate: Henry M. Emmons, Boston, 354 Commonwealth Avenue.)

Worcester North: C. Bertram Gay, Fitchburg, 62 Day Street. (Alternate: Francis A. Reynolds, Athol, 43 Cottage Street.)

Committee on Public Relations — Established 1931 (one councilor elected yearly by each district medical society; the president and president-elect of the Society are chairman and vice-chairman, respectively, and the vice-president and secretary of the Society are members *ex-officiis*).

Barnstable: William D. Kinney, Osterville.

Berkshire: Patrick J. Sullivan, Dalton, 471 Main Street.

Bristol North: James H. Brewster, Attleboro, 178 South Main Street.

Bristol South: Harold E. Perry, New Bedford, 159 Cottage Street.

Essex North: Harold R. Kurth, Lawrence, 57 Jackson Street.

Essex South: Loring Grimes, Swampscott, 84 Humphrey Street.

FRANKLIN: William J. Pelletier, Turners Falls, 113 Avenue A.
HAMPDEN: Patrick E. Gear, Holyoke, 188 Chestnut Street.
HAMPSHIRE: Alfred J. Bonneville, Hatfield, 60 Main Street.
MIDDLESEX EAST: J. Harper Blaisdell, Winchester. Office, Boston, 45 Bay State Road.
MIDDLESEX NORTH: Daniel J. Ellison, Lowell, 8 Merrimack Street.
MIDDLESEX SOUTH: John P. Nelligan, Cambridge, 2336 Massachusetts Avenue.
NORFOLK: Norman A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Avenue.
NORFOLK SOUTH: Fred A. Bartlett, Wollaston, 308 Beale Street.
PLYMOUTH: Charles D. McCann, Brockton, 12 Cottage Street.
SUFFOLK: Albert A. Hornor, Boston, 319 Longwood Avenue. (Secretary.)
WORCESTER: John Fallon, Worcester, 390 Main Street.
WORCESTER NORTH: James V. McHugh, Leominster, 100 Main Street.

Committee on Legislation — Established 1942 (one councilor elected yearly by each district medical society).

BARNSTABLE: Julius G. Kelley, Pocasset, Barnstable County Sanatorium.
BERKSHIRE: Clement F. Kernan, Pittsfield, 184 North Street.
BRISTOL NORTH: Ralph M. Chambers, Taunton, Taunton State Hospital.
BRISTOL SOUTH: Edwin D. Gardner, New Bedford, 150 Cottage Street.
ESSEX NORTH: Edward H. Ganley, Methuen, 251 Broadway.
ESSEX SOUTH: Charles A. Worthen, Lynn, 19 Park Street.
FRANKLIN: Howard M. Kemp, Greenfield, 42 Franklin Street.
HAMPDEN: William A. R. Chapin, Springfield, 121 Chestnut Street.
HAMPSHIRE: Arthur N. Ball, Northampton, Northampton State Hospital.
MIDDLESEX EAST: John M. Wilcox, Woburn, 6 Bennett Street.
MIDDLESEX NORTH: Archibald R. Gardner, Lowell, 16 Shattuck Street.
MIDDLESEX SOUTH: Brainard F. Conley, Malden, 51 Main Street (Chairman).
NORFOLK: Humphrey L. McCarthy, West Roxbury. Office Boston, 479 Beacon Street.
NORFOLK SOUTH: David L. Belding, Hingham. Office, Boston, 80 East Concord Street.
PLYMOUTH: John J. McNamara, Brockton, 231 Main Street.
SUFFOLK: William E. Browne, Boston, 587 Beacon Street.
WORCESTER: Lester M. Felton, Worcester, 36 Pleasant Street.
WORCESTER NORTH: Francis A. Reynolds, Athol, 43 Cottage Street.

Committee on Nominations — Established 1874 (one councilor and alternate elected yearly by each district medical society).

BARNSTABLE: William D. Kinney, Osterville. (Alternate: Paul M. Butterfield, Harwich.)
BERKSHIRE: Patrick J. Sullivan, Dalton, 471 Main Street. (Alternate: Charles F. Fasce, Pittsfield, 311 North Street.)
BRISTOL NORTH: William H. Allen, Mansfield, 70 North Main Street. (Alternate: Joseph L. Murphy, Taunton, 23 Cedar Street.)
BRISTOL SOUTH: Edmond F. Cody, New Bedford, 105 South 6th Street. (Alternate: Richard B. Butler, Fall River, 278 North Main Street.)
ESSEX NORTH: Guy L. Richardson, Haverhill, 94 Emerson Street. (Alternate: Rolf C. Norris, Methuen, 247 Broadway.)
ESSEX SOUTH: Paul E. Tivnan, Salem, 70 Washington Street. (Alternate: Peer P. Johnson, Beverly, 1 Monument Square.)
FRANKLIN: Howard M. Kemp, Greenfield, 42 Franklin Street. (Alternate: William J. Pelletier, Turners Falls, 113 Avenue A.)
HAMPDEN: Allen G. Rice, Springfield, 146 Chestnut Street. (Alternate: to be appointed.)
HAMPSHIRE: L. Beverly Pond, Easthampton, 115 Main Street. (Alternate: Joseph D. Collins, Northampton, 187 Main Street.)
MIDDLESEX EAST: Ralph R. Stratton, Melrose, 538 Lynn Fells Parkway. (Alternate: Edward M. Halligan, Reading, 37 Salem Street.)
MIDDLESEX NORTH: Herbert M. Larrabee, Tewksbury. Office, Lowell, 9 Central Street. (Alternate: William M. Collins, Lowell, 174 Central Street.)
MIDDLESEX SOUTH: Dwight O'Hara, Waltham. Office, Boston, 416 Huntington Avenue. (Alternate: Joseph C. Merriam, Framingham, 198 Union Avenue.)
NORFOLK: David D. Scannell, Jamaica Plain. Office, Boston, 475 Commonwealth Avenue. (Alternate: Harry J. Inglis, Chestnut Hill. Office, Boston, 43 Bay State Road.)
NORFOLK SOUTH: Daniel B. Reardon, Quincy, 1186 Hancock Street. (Alternate: James E. Knowlton, Wollaston, 579 Hancock Streeet.)
PLYMOUTH: Walter H. Pulsifer, Whitman, 26 Park Avenue. (Alternate: Bradford H. Peirce, South Hanson, Plymouth County Hospital.)
SUFFOLK: William B. Breed, Boston, 264 Beacon Street. (Alternate: Albert A. Hornor, Boston, 319 Longwood Avenue.)
WORCESTER: Royal P. Watkins, Worcester, 332 Main Street. (Alternate: William F. Lynch, Worcester, 390 Main Street.)
WORCESTER NORTH: Bartholomew P. Sweeney, Leominster, 5 Gardner Place. (Alternate: Francis A. Reynolds, Athol, 43 Cottage Street.)

STANDING COMMITTEES FOR 1944–1945

ELECTED BY THE COUNCIL, May 22, 1944

	Date of Appointment
PUBLICATIONS — Established 1825.	
Richard M. Smith	June 6, 1933 (appointed chairman May 21, 1941)
James P. O'Hare	June 9, 1936
Conrad Wesselhoeft	June 2, 1937
William B. Breed	February 7, 1940
Oliver Cope	May 21, 1941
ARRANGEMENTS — Established 1849.	
Roy J. Heffernan	May 25, 1942 (appointed chairman May 22, 1944)
Sidney C. Wiggin	June 24, 1942
G. Guy Bailey	November 13, 1942
Harold G. Giddings	May 22, 1944
Robert L. Goodale	May 22, 1944
ETHICS AND DISCIPLINE — Established 1871.	
Ralph R. Stratton	June 9, 1936 (appointed chairman May 21, 1941)
William J. Brickley	February 3, 1937
Allen G. Rice	June 1, 1938
Fred R. Jouett	May 21, 1940
Archibald R. Gardner	May 21, 1941
MEDICAL EDUCATION — Established 1881.	
Robert T. Monroe	May 21, 1941 (appointed chairman February 4, 1942)
George D. Henderson	June 1, 1938
Chester S. Keefer	February 4, 1942
Isaac R. Jankelson	May 25, 1942
Arthur W. Allen	May 24, 1943

MEMBERSHIP — Established 1897.

Harlan F. Newton	June 9, 1931 (appointed chairman May 25, 1942)
John E. Fish	June 17, 1930
Peirce H. Leavitt	June 1, 1938
Sumner H. Remick	May 24, 1943
Samuel N. Vose	March 15, 1944

William H. Allen, H. Quimby Gallupe, Albert E. Parkhurst — representing the Supervising Censors.

PUBLIC HEALTH — Established 1912.

Roy J. Ward	May 22, 1944 (chairman)
Edward G. Huber	May 22, 1944
Ernest M. Morris	May 22, 1944
Dwight O'Hara	May 22. 1944
Conrad Wesselhoeft (interim appointment)	July 27, 1944

MEDICAL DEFENSE — Established 1927.

Arthur W. Allen	June 7, 1927 (appointed chairman June 7, 1939)
Edwin D. Gardner	June 7, 1927
William R. Morrison (sec. *pro tem*)	June 9, 1936
Horatio Rogers	June 7, 1939
Ira M. Dixson	August 17, 1942

SOCIETY HEADQUARTERS — Established 1942.

J. Harper Blaisdell	May 22, 1944 (chairman)
Charles G. Mixter	June 8, 1942
Michael A. Tighe	May 24, 1943
Frank R. Ober	May 22, 1944
Daniel B. Reardon	May 22, 1944

FINANCE — Established 1938.

Francis C. Hall	July 8, 1943 (chairman)
Ernest L. Hunt	June 2, 1938
Charles F. Wilinsky	June 2, 1938
Edward J. O'Brien, Jr.	June 2, 1938
Peer P. Johnson	October 4, 1939

INDUSTRIAL HEALTH — Established 1942.

Dwight O'Hara	May 25, 1942 (chairman)
Thomas L. Shipman	May 25, 1942 (appointed vice-chairman May 22, 1944)
Joseph C. Aub	May 25, 1942
Daniel L. Lynch	May 25, 1942
John R. Agnew	May 22, 1944
Louis R. Daniels	May 22, 1944
John G. Downing	May 22, 1944

ADVISORY COMMITTEE TO COMMITTEE ON INDUSTRIAL HEALTH — Established 1942.

Manfred Bowditch, Alton S. Pope, Irving R. Tabershaw.

SPECIAL COMMITTEES

COMMITTEE ON CANCER — Established 1917.

George A. Moore (Brockton), *chairman*; Franklin G. Balch, Sr., Ernest M. Daland, Channing C. Simmons, Thomas J. Anglem.

REPRESENTATIVES TO THE MASSACHUSETTS CENTRAL HEALTH COUNCIL.

William D. Kinney, George D. Henderson, Robert B. Osgood, Merrill E. Champion, Roy J. Ward.

COMMITTEE ON POSTGRADUATE INSTRUCTION — Established 1932.

Reginald Fitz, *chairman*; Leroy E. Parkins, *secretary*; Joseph W. O'Connor, Robert N. Nye, Charles J. Kickham.

COMMITTEE ON PHYSICAL THERAPY — Established 1935.

Arthur L. Watkins, *chairman*, Franklin P. Lowry, Robert B. Osgood.

COMMITTEE TO CONSIDER EXPERT TESTIMONY — Established 1936.

Frank R. Ober, *chairman*; David Cheever, Francis P. McCarthy, Carl Bearse, William J. Brickley.

TWENTY-FIVE VOTING MEMBERS IN MASSACHUSETTS HOSPITAL SERVICE, INC. — Established 1939.

Benjamin H. Alton, Gerardo M. Balboni, William B. Breed, Laurence D. Chapin, Lucien R. Chaput, Hilbert F. Day, Augustus W. Dudley, John Fallon, George K. Fenn, Joseph E. Flynn, Archibald R. Gardner, Henry W. Godfrey, John H. Lambert, Alexander A. Levi, Joseph C. Merriam, Donald Munro, Albert E. Parkhurst, Helen S. Pittman, Allen G. Rice, Arthur T. Ronan, Frank W. Snow, George L. Steele, Ralph R. Stratton, John E. Talbot, Edward L. Young.

COMMITTEE CONCERNED WITH PREPAYMENT MEDICAL-CARE COSTS INSURANCE — Established 1940.

James C. McCann, *chairman*; Peirce H. Leavitt, Joseph C. Merriam, J. Harper Blaisdell, Merrill C. Sosman.

COMMITTEE ON TAX-SUPPORTED MEDICAL CARE — Established 1940.

John J. Dumphy, *chairman*; Albert A. Hornor, William J. Pelletier, Frederick S. Hopkins, Frank W. Snow.

COMMITTEE TO MEET WITH THE MASSACHUSETTS HOSPITAL ASSOCIATION — Established 1940.

Walter G. Phippen, *chairman*; Frederic Hagler, Edwin D. Gardner, Albert E. Parkhurst, Edward A. Adams, John Fallon.

COMMITTEE ON MATERNAL WELFARE — Established 1941.

Louis E. Phaneuf, *chairman*; Richard M. Smith, *vice-chairman*; Thomas Almy, Robert L. DeNormandie, Arthur F. G. Edgelow, Joseph W. O'Connor, Raymond S. Titus, Richard J. Williams, Warren R. Sisson, James M. Baty, Stewart H. Clifford, Arthur M. Kimberly, Florence L. McKay, Robert T. Moulton, Daniel J. Ellison, Ralph E. Cole (interim appointment), Benjamin deF. Lambert (interim appointment).

COMMITTEE ON REHABILITATION — Established 1941.

William E. Browne, *chairman*; William M. Collins, James J. Regan, Benjamin F. Andrews, Ralph M. Chambers, Arthur L. Watkins, John Fallon.

COMMITTEE ON POSTPAYMENT MEDICAL CARE — Established 1942.

Daniel J. Ellison, *chairman*; Harold F. Rowley, Charles F. Fasce, James H. Brewster, Harold E. Perry, Lucien R. Chaput, Loring Grimes, Howard M. Kemp, Patrick E. Gear, Alfred J. Bonneville, Wilfred L. McKenzie, Egon E. Kattwinkel, Norman A. Welch, Daniel B. Reardon (interim appointment), Michael F. Barrett, Francis T. Jantzen, James T. Brosnan, William G. LeBrecht.

MILITARY POSTGRADUATE COMMITTEE — Established 1942.

W. Richard Ohler, *chairman*; Leroy E. Parkins, *secretary*; Chester S. Keefer, Samuel H. Proger, Frank R. Ober, Gordon M. Morrison.

WAR PARTICIPATION COMMITTEE — Established 1943.

Guy L. Richardson, *chairman*; Ralph R. Stratton, Dwight O'Hara, Michael A. Tighe, Carl Bearse, Harold G. Giddings.

MEDICAL ADVISORY COMMITTEE TO REGIONAL OPA — Established 1943.

Joseph Garland, *chairman*; F. Gorham Brigham, Franklin W. White, Loring Grimes (interim appointment).

POSTWAR LOAN FUND COMMITTEE — Established 1943.

George L. Schadt, *chairman*; Edward P. Bagg, C. Sidney Burwell, John Homans, Eliot Hubbard, Jr., Peirce H. Leavitt, Herbert L. Lombard, Hyman Morrison, Walter G. Phippen, William F. Ryan, David D. Scannell, Charles A. Sparrow, Ralph R. Stratton, Michael A. Tighe, Charles F. Wilinsky.

COMMITTEE TO LOOK INTO THE POSSIBILITY OF BETTER PUBLICITY FOR THE MASSACHUSETTS MEDICAL SOCIETY — Established 1943.

Albert A. Hornor, *chairman*; Michael A. Tighe, Robert N. Nye, Norman A. Welch, Walter H. Pulsifer.

COMMITTEE ON MEDICAL INFORMATION BUREAU — Established 1944.

Walter G. Phippen, *chairman*; Michael A. Tighe, Arthur W. Allen.

REPRESENTATIVE TO MENTAL HEALTH FOR DEFENSE ORGANIZATION

Abraham Myerson.

REPRESENTATIVE TO THE HOSPITAL COUNCIL OF BOSTON FOR THE YEAR 1944.

William E. Browne.

REPRESENTATIVE ON THE MASSACHUSETTS COMMITTEE FOR NURSES PROCUREMENT AND ASSIGNMENT SERVICE.

Dwight O'Hara.

REPRESENTATIVE ON THE LEGISLATIVE COMMITTEE OF THE MASSACHUSETTS CENTRAL HEALTH COUNCIL.

William E. Browne.

DELEGATES AND ALTERNATES TO THE HOUSE OF DELEGATES, AMERICAN MEDICAL ASSOCIATION FOR 1944–1945

DELEGATES	ALTERNATES
June 1, 1943, to June 1, 1945	
Allen G. Rice, Springfield	Patrick J. Sullivan, Dalton
Richard H. Miller, Boston	John Fallon, Worcester
June 1, 1944, to June 1, 1946	
David D. Scannell, Jamaica Plain	Elmer S. Bagnall, Groveland
Dwight O'Hara, Waltham	Ernest L. Hunt, Worcester
Charles E. Mongan, Somerville	Charles J. Kickham, Brookline
Walter G. Phippen, Salem	John I. B. Vail, Hyannis

MASSACHUSETTS MEDICAL SERVICE

Officers and Directors

James C. McCann, M.D., president
Philip M. Morgan, vice-president
Edmund L. Twomey, treasurer
J. Harper Blaisdell, M.D.
Daniel J. Boyle
Thomas G. Brown
Thomas G. Dignan
J. H. Humphrey
Ernest A. Johnson
Harold B. Leland
Charles E. Mongan, M.D.
Frank R. Ober, M.D.
P. A. O'Connell
Roswell F. Phelps
Oliver G. Pratt
Samuel A. Robins, M.D.
R. F. Cahalane, executive director, 230 Congress Street, Boston

Members of the Corporation

William H. Allen, M.D.
Elmer S. Bagnall, M. D.
William A. R. Chapin, M.D.
Isaac S. F. Dodd, M.D.
Reginald Fitz, M.D.
Edwin D. Gardner, M.D.
C. Bertram Gay, M.D.
Loring Grimes, M.D.
Edward M. Halligan, M.D.
Eliot Hubbard, Jr., M.D.
Charles J. Kickham, M.D.
William D. Kinney, M.D.
Peirce H. Leavitt, M.D.
Donald Munro, M.D.
Dwight O'Hara, M.D.
William J. Pelletier, M.D.
Ralph S. Perkins, M.D.
L. Beverly Pond, M.D.
Sumner H. Remick, M.D.
Henry A. Robinson, M.D.
William F. Ryan, M.D.
Frank W. Snow, M.D.
Michael A. Tighe, M.D.

COUNCILORS FOR 1944–1945

(ELECTED BY THE DISTRICT MEDICAL SOCIETIES AT THEIR ANNUAL MEETINGS, APRIL 15 TO MAY 15, 1944.)

BARNSTABLE

J. N. Kelly, Orleans, River Rd., V. P.
P. M. Butterfield, Harwich, A. M. N. C.
C. H. Keene, Chatham, Seaview St.
J. G. Kelley, Pocasset, Barnstable County Sanatorium, Sec.
W. D. Kinney, Osterville, E. C., M. N. C.

BERKSHIRE

F. R. Smith, Pittsfield, 18 Bank Row, V. P.
N. N. Copeland, Pittsfield, 131 North St., Sec.
I. S. F. Dodd, Pittsfield, 34 Fenn St., E. C.
C. F. Fasce, Pittsfield, 311 North St., A. M. N. C.
C. F. Kernan, Pittsfield, 184 North St.
C. T. Leslie, Pittsfield, 18 Bank Row.
J. F. McLaughlin, Adams, 25 Park St.
Solomon Schwager, Pittsfield, 246 North St.
P. J. Sullivan, Dalton, 471 Main St., A. E. C., M. N. C.

BRISTOL NORTH

J. L. Murphy, Taunton, 23 Cedar St., V. P., A. M. N. C.
W. H. Allen, Mansfield, 70 North Main St., E. C., M. N. C.
J. H. Brewster, Attleboro, 178 South Main St.
R. M. Chambers, Taunton, Taunton State Hospital, A. E. C.
W. J. Morse, Attleboro, 34 Sanford St., Sec.
W. M. Stobbs, Attleboro, 63 Bank St.

BRISTOL SOUTH

Russell Wood, New Bedford, 160 William St., V. P.
G. W. Blood, Fall River, 82 New Boston Rd.
R. B. Butler, Fall River, 278 North Main St., A. E. C. A. M. N. C.
E. F. Cody, New Bedford, 105 South Sixth St., M. N. C.
J. A. Fournier, Fall River, 11 Choate St.
E. D. Gardner, New Bedford, 150 Cottage St., E. C.
F. M. Howes, New Bedford, 135 Cottage St.
H. E. Perry, New Bedford, 159 Cottage St.
R. H. Smith, Edgartown, North Water St.
A. H. Sterns, New Bedford, 31 Seventh St., Sec.
I. N. Tilden, Mattapoisett, Barstow St.
C. C. Tripp, New Bedford, 416 County St.
P. E. Truesdale, Fall River, 151 Rock St.

ESSEX NORTH

J. T. Batal, Andover, Office Lawrence, 281 Haverhill St., V. P.
E. S. Bagnall, Groveland, 281 Main St., President.
R. V. Baketel, Methuen, 7 Hampshire St.
G. J. Connor, Haverhill, 81 Merrimack St.
Elizabeth Councilman, Newburyport, 83 High St.
H. A. Fenton, Lawrence, 36 Winthrop Ave.
E. H. Ganley, Methuen, 251 Broadway
H. R. Kurth, Lawrence, 57 Jackson St., Sec.
P. J. Look, Andover, 115 Main St.
R. J. Neil, Methuen, 255 Broadway.

R. C. Norris, Methuen, 247 Broadway, A. E. C., A. M. N. C.
G. L. Richardson, Haverhill, 94 Emerson St., M. N. C.
F. W. Snow, Newburyport, 24 Essex St., E. C.
C. F. Warren, Amesbury, 1 School St.

ESSEX SOUTH

E. D. Reynolds, Danvers, 48 High St., V. P.
Bernard Appel, Lynn, 281 Ocean St.
H. A. Boyle, Middleton, Essex Sanatorium.
D. S. Clark, Salem, 2 Oliver St.
C. L. Curtis, Salem, 10 Federal St., A. E. C.
R. E. Foss, Peabody, 125 Main St.
Loring Grimes, Swampscott, 84 Humphrey St., E. C.
P. P. Johnson, Beverly, 1 Monument Sq., A. M. N. C.
H. M. Lowd, Swampscott, 90 Burrill St.
B. B. Mansfield, Ipswich, 4 Green St.
A. E. Parkhurst, Beverly, Monument Sq.
O. S. Pettingill, Middleton, Essex Sanatorium.
W. G. Phippen, Salem, 31 Chestnut St., Ex-Pres.
G. S. Rust, Gloucester, 48 Pleasant St.
H. D. Stebbins, Salem, 26 Chestnut St., Sec.
P. E. Tivnan, Salem, 70 Washington St., M. N. C.
J. W. Trask, East Lynn, 90 Ocean St.
C. F. Twomey, East Lynn, 80 Ocean St.
C. A. Worthen, Lynn, 19 Park St.

FRANKLIN

K. H. Rice, South Deerfield, 141 Main St., V. P.
H. L. Craft, Ashfield, Sec.
H. M. Kemp, Greenfield, 42 Franklin St., M. N. C.
M. B. Low, Deerfield, 31 Federal St., A. E. C.
W. J. Pelletier, Turners Falls, 113 Ave. A., E. C., A. M. N. C.

HAMPDEN

J. E. Dwyer, Springfield, 146 Chestnut St., V. P.
F. H. Allen, Holyoke, 16 Fairfield St.
E. P. Bagg, Holyoke, 207 Elm St., A. E. C.
J. M. Birnie, Springfield, 146 Chesnut St., Ex-Pres.
H. F. Byrnes, Springfield, 6 Chestnut St.
W. A. R. Chapin, Springfield, 121 Chestnut St., E. C.
J. L. Chereskin, Springfield, 333 Bridge St.
A. J. Douglas, Westfield, 93 Elm St.
E. C. Dubois, Springfield, 174 Buckingham St.
G. L. Gabler, Holyoke, 4 Bullard Ave.
P. E. Gear, Holyoke, 188 Chestnut St.
Frederic Hagler, Springfield, 20 Maple St.
G. D. Henderson, Holyoke, 176 Chestnut St.
F. S. Hopkins, Springfield, 146 Chestnut St.
Charles Jurist, Springfield, 70 Chestnut St.
M. W. Pearson, Ware, 19 Pleasant St.
A. G. Rice, Springfield, 146 Chestnut St., M. N. C.
G. L. Schadt, Springfield, 44 Chestnut St., Ex-Pres.
J. A. Seaman, Longmeadow, Office Springfield, 20 Maple St.
G. C. Steele, West Springfield, 39 Church St., Sec.
G. L. Steele, Springfield, 20 Maple St.

HAMPSHIRE

A. N. Ball, Northampton, State Hospital, V. P.
A. J. Bonneville, Hatfield, 60 Main St.
J. D. Collins, Northampton, 187 Main St., A. E. C., A. M. N. C.
W. M. Dobson, Northampton, Veterans Administration Facility.
L. B. Pond, Easthampton, 115 Main St., E. C., M. N. C.
Mary P. Snook, Chesterfield, Sec.

MIDDLESEX EAST

J. H. Kerrigan, Stoneham, 481 Main St. V. P.
J. H. Blaisdell, Winchester, Office Boston, 45 Bay State Rd., C.
R. M. Burgoyne, Winchester, 15 Washington St.
Richard Dutton, Wakefield, 33 Avon St., A. E. C.
E. M. Halligan, Reading, 37 Salem St., E. C., A. M. N. C.
R. W. Layton, Melrose, 8 Porter St., Sec.
M. J. Quinn, Winchester, 44 Church St.
R. R. Stratton, Melrose, 538 Lynn Fells Parkway, M. N. C., C.
J. M. Wilcox, Woburn, 6 Bennett St.

MIDDLESEX NORTH

J. J. Cassidy, Lowell, 9 Central St., V. P.
H. R. Coburn, Lowell, 202 Merrimack St.
W. M. Collins, Lowell, 174 Central St., A. E. C., A. M. N. C.
D. J. Ellison, Lowell, 8 Merrimack St.
A. R. Gardner, Lowell, 16 Shattuck St.
H. M. Larrabee, Lowell, 9 Central St., M. N. C.
B. D. Leahey, Lowell, 9 Central St., Sec.
W. F. Ryan, Lowell, 219 Central St., E. C.
M. A. Tighe, Lowell, 9 Central St., Secretary.

MIDDLESEX SOUTH

H. G. Giddings, Newton Centre, Office Boston, 270 Commonwealth Ave., V. P.
C. F. Atwood, Arlington, 821 Massachusetts Ave.
E. W. Barron, Malden, Office Boston, 20 Ash St.
W. B. Bartlett, Concord, 28 Monument St.
Harris Bass, Everett, 351 Broadway.
J. M. Baty, Belmont, Office Brookline, 1101 Beacon St.
J. D. Bennett, West Somerville, 72 College Ave.
E. H. Bigelow, Framingham, Hotel Kendall, Ex-Pres.
W. O. Blanchard, Newton, 465 Centre St.
G. F. H. Bowers, Newton Highlands, 156 Woodward St.
Madelaine R. Brown, Cambridge, Office Boston, 264 Beacon St.
R. W. Buck, Waban, Office Boston, 5 Bay State Rd.
E. J. Butler, Cambridge, 25 Garden St.
J. F. Casey, Allston, Office Boston, 475 Commonwealth Avenue.
C. W. Clark, Newtonville, 363 Walnut St.
B. F. Conley, Malden, 51 Main St.
J. A. Daley, Natick, 36 Pond St. (interim appointment).
H. F. Day, Cambridge, 34 Kirkland St.
C. L. Derick, Newton Highlands, Office Boston, 412 Beacon St.
J. G. Downing, Newton, Office Boston, 520 Commonwealth Ave.
R. A. Drake, West Medford, 298 High Street.
C. W. Finnerty, West Somerville, 5 Pearson Rd.
H. Q. Gallupe, Waltham, 751 Main St.
Stanton Garfield, Concord, 20 Sudbury St.
F. W. Gay, Malden, 20 Park St.
H. W. Godfrey, Auburndale, 14 Hancock St.
J. L. Golden, Medford, 86 Forest St.
A. D. Guthrie, Medford, 408 Salem St.
Eliot Hubbard, Jr., Cambridge, 29 Highland St., Treasurer.
A. M. Jackson, Everett, 512 Broadway, A. E. C.
F. R. Jouett, Cambridge, 1 Craigie St.
E. E. Kattwinkel, West Newton, 65 Sterling St.
A. A. Levi, Newton, Office Boston, 481 Beacon St., Sec.
F. P. Lowry, Newton, 313 Washington St.
A. N. Makechnie, Cambridge, 14 Upland Rd.
P. H. Means, Cambridge, 1 Waterhouse St.
J. C. Merriam, Framingham, 198 Union Ave., A. M. N. C.
C. E. Mongan, Somerville, 24 Central St., Ex-Pres.
J. P. Nelligan, Cambridge, 2336 Massachusetts Ave.
E. J. O'Brien, Jr., Newton, Office Boston, 270 Commonwealth Ave.
Dwight O'Hara, Waltham, Office Boston, 416 Huntington Ave., E. C., M. N. C., C.
Fabyan Packard, Belmont, Office Boston, Soldiers' Field.
L. G. Paul, Newton Centre, Office Boston, 270 Commonwealth Ave.
T. E. Reilly, Marlboro, 6 Newton St.
S. H. Remick, Waltham, 735 Trapelo Rd., Vice-President
Max Ritvo, Newton, Office Boston, 416 Marlboro St.
E. H. Robbins, Somerville, 334 Broadway.
E. S. A. Robinson, Newton Centre, Office Jamaica Plain, 375 South St.
M. J. Schlesinger, Newton, Office Boston, 330 Brookline Ave.
E. W. Small, Belmont, 68 Leonard St.
H. P. Stevens, Cambridge, 1 Craigie St.
H. W. Thayer, Newtonville, 355 Walnut St.
K. J. Tillotson, Waverley, McLean Hospital (interim appointment).
A. B. Toppan, Watertown, 289 Mt. Auburn St.

J. E. Vance, Natick, Office Boston, 29 Bay State Rd.
Fresenius Van Nüys, Weston, 338 Boston Post Rd.
C. F. Walcott, Cambridge, 81 Sparks St.
A. L. Watkins, Arlington, Office Boston, Massachusetts General Hospital.
B. M. Wein, Newton, Office Boston, 471 Commonwealth Ave.
B. S. Wood, Weston, Office Waltham, 751 Main St.
Alfred Worcester, Waltham, 314 Bacon St., Ex-Pres.
Hovhannes Zovickian, Watertown, 528 Mt. Auburn St.

NORFOLK

Albert Ehrenfried, Brookline, Office Boston, 520 Beacon St., V. P.
J. R. Barry, West Roxbury, 1857 Centre St.
Carl Bearse, Boston, 483 Beacon St.
Arthur Berk, Brookline, Office Boston, 270 Commonwealth Ave.
M. I. Berman, Dorchester, 1071 A Blue Hill Ave.
G. F. Blood, Roslindale, 20 Belgrade Ave.
J. E. Burns, Wellesley Hills, Office Boston, 475 Commonwealth Ave.
J. H. Carey, Dorchester, 103 Melville Ave.
D. J. Collins, Norwood, 100 Day St.
William Dameshek, Brookline, Office Boston, 371 Commonwealth Ave.
G. L. Doherty, West Roxbury, Office Boston, 466 Commonwealth Ave.
H. M. Emmons, Needham, Office Boston, 354 Commonwealth Ave., A. E. C.
J. C. V. Fisher, West Roxbury, 1811 Centre St.
Susannah Friedman, Roxbury, Office Boston, 485 Commonwealth Ave.
B. A. Godvin, Jamaica Plain, Office Boston, 483 Beacon St.
D. C. Goldfarb, Brookline, Office Boston, 483 Beacon St.
J. B. Hall, Roxbury, 60 Windsor St.
H. B. Harris, East Milton, Office Dorchester, 487 Columbia Rd.
R. J. Heffernan, Jamaica Plain, Office Brookline, 1101 Beacon St., C.
H. J. Inglis, Chestnut Hill, Office Boston, 43 Bay State Rd., A. M. N. C.
P. J. Jakmauh, Milton, Office South Boston, 509 Broadway.
I. R. Jankelson, Jamaica Plain, Office Boston, 483 Beacon St.
C. J. Kickham, Brookline, Office Boston, 524 Commonwealth Ave., E. C.
C. J. E. Kickham, Jamaica Plain, Office Boston, 12 Bay State Rd., Sec.
E. L. Kickham, Brookline, Office Boston, 270 Commonwealth Ave.
H. M. Landesman, Roxbury, Office Boston, 429 Marlborough St.
Timothy Leary, Jamaica Plain, Office Boston, 784 Massachusetts Ave.
D. S. Luce, Canton, 553 Washington St.
C. M. Lydon, Dorchester, 276 Bowdoin St.
D. L. Lynch, Roslindale, Office Boston, 245 State St.
F. P. McCarthy, Milton, Office Boston, 371 Commonwealth Ave.
H. L. McCarthy, West Roxbury, Office Boston, 479 Beacon St.
K. T. Monroe, Brookline, Office Boston, 270 Commonwealth Ave., C.
F. J. Moran, Dedham, 395 Washington St.
Hyman Morrison, Roxbury, Office Boston, 483 Beacon St.
D. J. Mullane, Jamaica Plain, 776 Centre St.
Abraham Myerson, Brookline, Office Boston, 475 Commonwealth Ave. (interim appointment).
M. W. O'Connell, West Roxbury, Office Boston, Boston City Hospital.
G. W. Papen, Brookline, Office Boston, 31 Milk St.
H. C. Petterson, West Roxbury, Office Boston, 29 Bay State Rd.
Frederick Reis, Jamaica Plain, Office Boston, 416 Huntington Ave.
S. A. Robins, Roxbury, Office Boston, 636 Beacon St.
D. D. Scannell, Jamaica Plain, Office Boston, 475 Commonwealth Ave., M. N. C.
J. A. Seth, Milton, Office Boston, 47 Bay State Rd.
Kathleyne S. Snow, Jamaica Plain, Office Boston, 466 Commonwealth Ave.
J. W. Spellman, Chestnut Hill, Office Brookline, 1101 Beacon St.
M. H. Spellman, Jamaica Plain, Office Boston, 475 Commonwealth Ave.
J. P. Treanor, Jr., Jamaica Plain, Office Brookline, 1101 Beacon St.
W. J. Walton, Dorchester, 106 Bowdoin St.
S. H. Weiner, Roxbury, Office Boston, 524 Commonwealth Ave.
N. A. Welch, West Roxbury, Office Boston, 520 Commonwealth Ave., Assistant Treasurer.

NORFOLK SOUTH

H. A. Robinson, Hingham, 205 North St., V. P., E. C.
C. S. Adams, Wollaston, 62 Brooks St.
F. A. Bartlett, Wollaston, 308 Beale St.
D. L. Belding, Hingham, Office Boston, 80 East Concord St.
Harry Braverman, Quincy, 43 School St.
Frederick Hinchcliffe, Cohasset, 117 South Main St.
E. K. Jenkins, South Braintree, Norfolk County Hospital, Sec.
J. E. Knowlton, Quincy, 579 Hancock St., A. M. N. C.
N. R. Pillsbury, South Braintree, Norfolk County Hospital, A. E. C.
D. B. Reardon, Quincy, 1186 Hancock St., M. N. C.

PLYMOUTH

P. H. Leavitt, Brockton, 129 West Elm St., V. P., E. C.
S. J. Beers, Plymouth, 118 Court St.
C. H. King, Duxbury, Cedar St.
C. D. McCann, Brockton, 12 Cottage St., A. E. C.
R. C. McLeod, Brockton, Goddard Hospital, Sec.
J. J. McNamara, Brockton, 231 Main St.
G. A. Moore, Brockton, 167 Newbury St.
B. H. Peirce, South Hanson, Plymouth County Hospital, A. M. N. C.
E. L. Perry, Middleboro, 39 Oak St.
W. H. Pulsifer, Whitman, 26 Park Ave., M. N. C.

SUFFOLK

Donald Munro, Boston, 818 Harrison Ave., V. P., E. C.
A. W. Allen, Boston, 266 Beacon St., C.
J. W. Bartol, Boston, 1 Chestnut St., Ex-Pres.
W. B. Breed, Boston, 264 Beacon St., M. N. C.
W. J. Brickley, Boston, 524 Commonwealth Ave.
W. E. Browne, Boston, 587 Beacon St.
W. B. Castle, Boston, Boston City Hospital.
David Cheever, Boston, 193 Marlborough St.
Pasquale Costanza, East Boston, 238 Maverick St.
N. W. Faxon, Boston, Massachusetts General Hospital.
G. B. Fenwick, Chelsea, 38 Cary Ave.
Jacob Fine, Boston, 330 Brookline Ave.
Reginald Fitz, Boston, 319 Longwood Ave., President-Elect.
Somers Fraser, Boston, 395 Commonwealth Ave.
Maurice Fremont-Smith, Boston, 12 Hereford St.
Channing Frothingham, Boston, Office Jamaica Plain, 1153 Centre St., Ex-Pres.
Joseph Garland, Boston, 266 Beacon St.
R. L. Goodale, Boston, 330 Dartmouth St., Sec.
F. C. Hall, Boston, 372 Marlborough St., C.
John Homans, Boston, 311 Beacon St. (interim appointment).
A. A. Hornor, Boston, 319 Longwood Ave., A. M. N. C.
L. M. Hurxthal, Boston, 605 Commonwealth Ave.
C. S. Keefer, Boston, 65 East Newton St.
H. A. Kelly, Winthrop, 200 Pleasant St.
R. I. Lee, Boston, 264 Beacon St., Ex-Pres.
C. C. Lund, Boston, 319 Longwood Ave., A. E. C.
W. J. Mixter, Boston, 319 Longwood Ave.
H. L. Musgrave, Revere, 622 Beach St.
H. F. Newton, Boston, 319 Longwood Ave., C.
R. N. Nye, Boston, 8 Fenway.
F. R. Ober, Boston, 234 Marlborough St., Ex-Pres.
F. W. O'Brien, Boston, 465 Beacon St.
J. P. O'Hare, Boston, 520 Commonwealth Ave.

L. E. Parkins, Boston, 12 Bay State Rd.
L. E. Phaneuf, Boston, 270 Commonwealth Ave.
Helen S. Pittman, Boston, 264 Beacon St.
J. H. Pratt, Boston, 30 Bennet St.
J. J. Regan, Boston, 520 Commonwealth Ave. (interim appointment).
W. H. Robey, Boston, 202 Commonwealth Ave., Ex-Pres.
H. F. Root, Boston, 81 Bay State Rd.
R. M. Smith, Boston, 330 Dartmouth St., C.
M. C. Sosman, Boston, 721 Huntington Ave.
E. F. Timmins, South Boston, 527 Broadway.
J. J. Todd, Boston, 479 Beacon St.
S. N. Vose, Boston, 29 Bay State Rd.
Conrad Wesselhoeft, Boston, 315 Marlborough St.
C. F. Wilinsky, Boston, 330 Brookline Ave.

WORCESTER

B. H. Alton, Worcester, 27 Elm St., V. P.
C. R. Abbott, Clinton, 60 Walnut St.
B. F. Andrews, Worcester, 36 Pleasant St.
A. W. Atwood, Worcester, 390 Main St.
George Ballantyne, Worcester, 27 Elm St.
Gordon Berry, Worcester, 36 Pleasant St., A. E. C.
W. P. Bowers, Clinton, 264 Chestnut St., Ex-Pres.
E. J. Crane, Holden, Armington Lane.
J. J. Dumphy, Worcester, 390 Main St.
John Fallon, Worcester, 390 Main St.
L. M. Felton, Worcester, 36 Pleasant St.
J. V. Gallagher, Milford, 224 Main St.
E. R. Leib, Worcester, 36 Pleasant St.
L. P. Leland, Worcester, 36 Pleasant St., Sec.
W. F. Lynch, Worcester, 390 Main St., A. M. N. C.
J. C. McCann, Worcester, 390 Main St.
A. E. O'Connell, Worcester, 390 Main St.
H. L. Paine, North Grafton, Grafton State Hospital
R. S. Perkins, Worcester, 10 Hackfeld Rd., E. C.
C. A. Sparrow, Worcester, 21 West St.
O. H. Stansfield, Worcester, 36 Pleasant St.
T. L. Story, Southbridge, 17 Maple St. (interim appointment).
J. C. Sullivan, Webster, 18 Negus St.
R. J. Ward, Worcester, 9 Bellevue St., C.
R. P. Watkins, Worcester, 332 Main St., M. N. C.
B. C. Wheeler, Worcester, 27 Elm St.
S. B. Woodward, Worcester, 58 Pearl St., Ex-Pres.

WORCESTER NORTH

H. D. Bone, Gardner, 19 Pleasant St., V. P.
E. A. Adams, Fitchburg, 44 Oliver St., Sec.
C. B. Gay, Fitchburg, 62 Day St., E. C.
G. P. Keaveny, Fitchburg, 62 Fox St.
J. V. McHugh, Leominster, 100 Main St.
F. A. Reynolds, Athol, 43 Cottage St., A. E. C., A. M. N. C.
B. P. Sweeney, Leominster, 5 Gardner Place, M. N. C.

The initials *E. C.* following the name of a councilor indicate that he is a member of the Executive Committee, and *A. E. C.* that he is an alternate member of the Executive Committee; *M. N. C.* that he is a member of the Committee on Nominations and *A. M. N. C.* that he is an alternate member of the Committee on Nominations; *V. P.* that a member is a councilor by virtue of his office as president of a district society and so vice-president of the general society; *C.* by virtue of his office as chairman of a standing committee; *Sec.* by virtue of his office as secretary of a district society and *Ex-Pres.* by virtue of being a past president.

CENSORS FOR 1944–1945

BARNSTABLE

W. D. Kinney, Osterville, *supervisor.*
E. F. Curry, Sagamore.
C. E. Harris, Hyannis.
J. I. B. Vail, Hyannis.
D. H. Hiebert, Provincetown.

BERKSHIRE

C. T. Leslie, Pittsfield, *supervisor.*
I. S. F. Dodd, Pittsfield
M. M. Brown, North Adams.
A. C. England, Pittsfield,
W. T. Frawley, Pittsfield.

BRISTOL NORTH

W. H. Allen, Mansfield, *supervisor.*
L. E. Butler, Taunton.
A. J. Leddy, Taunton.
J. H. Brewster, Attleboro.
W. M. Stobbs, Attleboro.

BRISTOL SOUTH

(Supervisor to be appointed.)
E. A. McCarthy, Fall River
F. M. Howes, New Bedford.
W. F. MacKnight, Fall River.
C. C. Persons, New Bedford.

ESSEX NORTH

R. V. Baketel, Methuen, *supervisor.*
L. C. Peirce, Newburyport.
E. M. Gale, Merrimac.
F. A. O'Reilly, Lawrence.
W. W. Ferrin, Haverhill.

ESSEX SOUTH

A. E. Parkhurst, Beverly, *supervisor.*
J. G. Adams, Salem.
W. C. Inman, Danvers.
R. A. Harpin, Lynn.
I. B. Hull, Gloucester.

FRANKLIN

W. J. Pelletier, Turners Falls, *supervisor.*
A. H. Ellis, Greenfield.
P. M. Freeman, Greenfield.
J. E. Moran, Greenfield.
F. W. Dean, Northfield.

HAMPDEN

Frederic Hagler, Springfield, *supervisor.*
J. M. Gilchrist, Springfield.
A. F. G. Edgelow, Springfield.
John Pallo, Westfield.
G. D. Henderson, Holyoke.

HAMPSHIRE

A. J. Bonneville, Hatfield, *supervisor.*
M. E. Cooney, Northampton.
T. F. Corriden, Northampton.
J. E. Hayes, Northampton.
C. H. Wheeler, Haydenville.

MIDDLESEX EAST

M. J. Quinn, Winchester, *supervisor.*
J. H. Fay, Melrose.
C. E. Montague, Wakefield.
S. H. Moses, Winchester.
C. R. Baisley, Reading.

MIDDLESEX NORTH

W. F. Ryan, Lowell, *supervisor.*
F. R. Brady, Lowell.
R. C. Stewart, Lowell.
H. L. Leland, Lowell.
J. D. Sweeney, Lowell.

MIDDLESEX SOUTH

H. Q. Gallupe, Waltham, *supervisor.*
E. H. Robbins, Somerville.
A. N. Makechnie, Cambridge.
J. E. Vance, Natick.
H. W. Thayer, Newtonville.

NORFOLK

Hyman Morrison, Roxbury, *supervisor.*
C. J. Kickham, Brookline.
C. E. Allard, Dorchester.
Saul Berman, Chestnut Hill.
Kathleyne S. Snow, Jamaica Plain.

NORFOLK SOUTH

C. S. Adams, Wollaston, *supervisor.*
D. L. Belding, Hingham.
W. L. Sargent, Quincy.
C. J. Lynch, Quincy.
R. R. Ryan, South Weymouth.

PLYMOUTH
E. L. Perry, Middleboro, *supervisor*.
J. H. Dunn, Rockland.
J. A. Pettey, Brockton.
D. W. Pope, Brockton.
W. T. Hanson, Bridgewater.

SUFFOLK
J. H. Pratt, Boston, *supervisor*.
H. T. Hutchins, Boston.
A. J. A. Campbell, Boston.
W. E. Browne, Boston.
E. F. Timmins, South Boston.

WORCESTER
B. C. Wheeler, Worcester, *supervisor*.
B. H. Flower, Shrewsbury.
George Ballantyne, Worcester.
R. S. Newton, Westboro.
H. N. Kelley, Worcester.

WORCESTER NORTH
C. B. Gay, Fitchburg, *supervisor*.
F. J. Djerf, Fitchburg.
G. P. Keaveny, Fitchburg.
J. A. McLean, Ayer.
F. A. Reynolds, Athol.

VICE-PRESIDENTS OF THE MASSACHUSETTS MEDICAL SOCIETY (*Ex-Officiis*) FOR 1944–1945

PRESIDENTS OF DISTRICT MEDICAL SOCIETIES

(Arranged according to seniority of fellowship in the Massachusetts Medical Society)

NORFOLK — Albert Ehrenfried, Brookline.
MIDDLESEX NORTH — James J. Cassidy, Lowell.
MIDDLESEX SOUTH — Harold G. Giddings, Newton Centre.
MIDDLESEX EAST — Joseph H. Kerrigan, Stoneham.
HAMPDEN — John E. Dwyer, Springfield.
HAMPSHIRE — Arthur N. Ball, Northampton.
BRISTOL NORTH — Joseph L. Murphy, Taunton.
NORFOLK SOUTH — Henry A. Robinson, Hingham.
PLYMOUTH — Peirce H. Leavitt, Brockton.
WORCESTER NORTH — Herman D. Bone, Gardner.
WORCESTER — Benjamin H. Alton, Worcester.
SUFFOLK — Donald Munro, Boston.
BRISTOL SOUTH — Russell Wood, New Bedford.
FRANKLIN — Kenneth H. Rice, South Deerfield.
ESSEX NORTH — John T. Batal, Lawrence.
BERKSHIRE — Floyd R. Smith, Pittsfield.
ESSEX SOUTH — Edwin D. Reynolds, Danvers.
BARNSTABLE — Joseph N. Kelly, Orleans.

COMMISSIONERS OF TRIAL FOR 1944–1945

BARNSTABLE — F. O. Cass, Provincetown.
BERKSHIRE — J. B. Thomes, Pittsfield.
BRISTOL NORTH — J. W. Cook, Mansfield.
BRISTOL SOUTH — A. C. Lewis, Fall River.
ESSEX NORTH — F. W. Anthony, Haverhill.
ESSEX SOUTH — O. C. Blair, Lynn.
FRANKLIN — K. W. D. Jacobus, Turners Falls.
HAMPDEN — F. K. Dutton, Springfield.
HAMPSHIRE — E. H. Copeland, Northampton.
MIDDLESEX EAST — W. H. Keleher, Woburn.
MIDDLESEX NORTH — J. F. Boyle, Lowell.
MIDDLESEX SOUTH — H. P. Stevens, Cambridge.
NORFOLK — W. J. Walton, Dorchester.
NORFOLK SOUTH — F. A. Bartlett, Wollaston.
PLYMOUTH — J. A. Carriuolo, Brockton.
SUFFOLK — J. R. Torbert, Boston.
WORCESTER — W. P. Bowers, Clinton.
WORCESTER NORTH — A. P. Lachance, Gardner.

OFFICERS OF THE SECTIONS FOR 1945

ELECTED BY THE SECTIONS

SECTION OF MEDICINE
Chairman, Albert A. Hornor, Boston; *vice-chairman*, Daniel J. Ellison, Lowell; *secretary*, Francis C. Hall, Boston.

SECTION OF SURGERY
Chairman, Charles F. Twomey, East Lynn; *secretary*, Alexander J. A. Campbell, Boston.
Executive Committee — Stanley J. G. Nowak, Belmont and Boston (1 year); Edward L. Young, Jr., Brookline and Boston (2 years); E. Parker Hayden, Brookline and Boston (3 years).

SECTION OF PEDIATRICS
Chairman, Floyd R. Smith, Pittsfield; *secretary*, Gerald N. Hoeffel, Cambridge.
Executive Committee — *Chairman*, Philip H. Sylvester, Boston; James Marvin Baty, Belmont and Brookline; Leroy T. Stokes, Haverhill.

SECTION OF OBSTETRICS AND GYNECOLOGY
Chairman, Arthur F. G. Edgelow, Springfield; *vice-chairman*, William J. McDonald, Boston; *secretary*, George Van S. Smith, Brookline.

SECTION OF RADIOLOGY
Chairman, George Levene, Chestnut Hill and Boston; secretary, Edward B. D. Neuhauser, Cambridge and Boston.

SECTION OF PHYSIOTHERAPY
Chairman, Arthur L. Watkins, Arlington and Boston; *secretary*, Howard Moore, Newton and Boston.

SECTION OF DERMATOLOGY AND SYPHILOLOGY
Chairman, Bernard Appel, Lynn; *secretary*, Fenner A. Chace, Fall River.

OFFICERS OF THE DISTRICT MEDICAL SOCIETIES FOR 1944–1945

BARNSTABLE — *President*, Joseph N. Kelly, Orleans; *vice-president*, Harold F. Rowley, Harwich Port; *secretary*, Julius G. Kelley, Pocasset; *treasurer*, Frank Travers, Barnstable; *librarian*, Carroll H. Keene, Chatham; *executive and public-relations councilor*, William D. Kinney, Osterville; *legislative councilor*, Julius G. Kelley, Pocasset.

BERKSHIRE — *President*, Floyd R. Smith, Pittsfield; *vice-president*, Thomas H. Nelligan, Pittsfield; *secretary*, N. Newall Copeland, Pittsfield; *treasurer*, Daniel N. Beers, Pittsfield; *executive councilor*, Isaac S. F. Dodd, Pittsfield; *legislative councilor*, Clement F. Kernan, Pittsfield; *public-relations councilor*, Patrick J. Sullivan, Dalton.

BRISTOL NORTH — *President*, Joseph L. Murphy, Taunton; *vice-president*, William M. Stobbs, Attleboro; *secretary*, William J. Morse, Attleboro; *treasurer*, Joseph V. Chatigny, Taunton; *executive councilor*, William H. Allen, Mansfield; *legislative councilor*, Ralph M. Chambers, Taunton; *public-relations councilor*, James H. Brewster, Attleboro.

BRISTOL SOUTH — *President*, Russell Wood, New Bedford; *vice-president*, John C. Corrigan, Fall River; *secretary and treasurer*, Albert H. Sterns, New Bedford; *executive and legislative councilor*, Edwin D. Gardner, New Bedford; *public-relations councilor*, Harold E. Perry, New Bedford.

ESSEX NORTH — *President*, John T. Batal, Lawrence; *vice-president*, Percy J. Look, Andover; *secretary*, Harold R. Kurth, Lawrence; *treasurer*, Guy L. Richardson, Haverhill; *auditor*, Paul Nettle, Bradford; *executive councilor*, Frank W. Snow, Newburyport; *legislative councilor*, Edward H. Ganley, Methuen; *public-relations councilor*, Harold R. Kurth, Lawrence.

ESSEX SOUTH — *President*, Edwin D. Reynolds, Danvers; *vice-president*, William R. Irving, Gloucester; *secretary*, Henry D. Stebbins, Marblehead; *treasurer*, Charles F. Deering, Danvers; *executive and public-relations councilor*, Loring Grimes, Swampscott; *legislative councilor*, Charles A. Worthen, Lynn.

FRANKLIN — *President*, Kenneth H. Rice, South Deerfield; *vice-president*, John B. Temple, Shelburne Falls; *secretary and treasurer*, Harry L. Craft, Ashfield; *executive and public-relations councilor*, William J. Pelletier, Turners Falls; *legislative councilor*, Howard M. Kemp, Greenfield.

HAMPDEN — *President*, John E. Dwyer, Springfield; *vice-president*, Merrill F. Hosmer, Longmeadow; *secretary and treasurer*, George C. Steele, West Springfield; *executive and legislative councilor*, William A. R. Chapin, Springfield; *public-relations councilor*, Patrick E. Gear, Holyoke.

HAMPSHIRE — *President*, Arthur N. Ball, Northampton; *vice-president*, Joseph D. Fallon, Northampton; *secretary and treasurer*, Mary P. Snook, Chesterfield; *librarian*, Abbie M. O'Keefe, Northampton; *executive councilor*, L. Beverly Pond, Easthampton; *legislative councilor*, Arthur N. Ball, Northampton; *public-relations councilor*, Alfred J. Bonneville, Hatfield.

MIDDLESEX EAST — *President*, Joseph H. Kerrigan, Stoneham; *vice-president*, Justin L. Anderson, Reading; *secretary*, Roy W. Layton, Melrose; *treasurer*, Albert E. Small, Melrose; *librarian*, Raymond E. Militzer, Woburn; *executive councilor*, Edward M. Halligan, Reading; *legislative councilor*, John M. Wilcox, Woburn; *public-relations councilor*, J. Harper Blaisdell, Winchester.

MIDDLESEX NORTH — *President*, James J. Cassidy, Lowell; *vice-president*, William I. Wiggin, Lowell; *secretary*, Brendan D. Leahey, Lowell; *treasurer*, Mason D. Bryant, Lowell; *executive councilor*, William F. Ryan, Lowell; *legislative councilor*, Archibald R. Gardner, Lowell; *public-relations councilor*, Daniel J. Ellison, Lowell.

MIDDLESEX SOUTH — *President*, Harold G. Giddings, Newton Centre; *vice-president*, Brainard F. Conley, Malden; *secretary*, Alexander A. Levi, Newton Centre; *treasurer*, Fabyan Packard, Belmont; *orator*, Varaztad H. Kazanjian, Belmont; *executive councilor*, Dwight O'Hara, Waltham; *legislative councilor*, Brainard F. Conley, Malden; *public-relations councilor*, John P. Nelligan, Cambridge.

NORFOLK — *President*, Albert Ehrenfried, Brookline; *vice-president*, Dean S. Luce, Canton; *secretary*, Charles J. E. Kickham, Jamaica Plain; *treasurer*, Frederick Reis, Jamaica Plain; *executive councilor*, Charles J. Kickham, Brookline; *legislative councilor*, Humphrey L. McCarthy, West Roxbury; *public-relations councilor*, Norman A. Welch, West Roxbury.

NORFOLK SOUTH — *President*, Henry A. Robinson, Hingham; *vice-president*, Frank W. Crawford, Holbrook; *secretary and librarian*, Ebenezer K. Jenkins, Braintree; *treasurer*, Charles Djerf, Quincy; *executive councilor*, Henry A. Robinson, Hingham; *legislative councilor*, David L. Belding, Hingham; *public-relations councilor*, Fred A. Bartlett, Wollaston.

PLYMOUTH — *President*, Peirce H. Leavitt, Brockton; *vice-president*, Paul B. Kelly, Plymouth; *secretary*, Ralph C. McLeod, Brockton; *treasurer*, Rudolph A. Kruger, Brockton; *librarian*, John H. Weller, Bridgewater; *executive councilor*, Peirce H. Leavitt, Brockton; *legislative councilor*, John J. McNamara, Brockton; *public-relations councilor*, Charles D. McCann, Brockton.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 30311

PRESENTATION OF CASE

A thirty-nine-year-old housewife was admitted to the hospital for study.

The patient had been in apparent good health until two years before entry when she experienced intermittent attacks of epigastric pain, following one year of "beer drinking and smoking almost incessantly." The pain was centered in the pit of the stomach and radiated to the right scapula. During the attacks she was unable "to sit or lie still or get her breath." Leaning forward and pressing against the stomach with a pillow brought relief. She was seen by her physician, who attributed her condition to drinking and prescribed some pills and a liquid, which gave some relief. The attacks recurred on an average of once a month and lasted for a few days. About nine months before entry the attacks became more frequent and more severe. At that time she had an episode of jaundice with chills, fever and pain that subsided spontaneously. Following this she had a severe attack of epigastric pain that radiated to the back and along both costal margins. The pain occurred after eating and was accompanied by copious vomiting. This episode lasted for a week, although she was temporarily relieved by medication. Eight months before entry she was admitted to a hospital in another city, where a history of intolerance for fatty and fried foods and a story of constipation for a year were elicited.

Physical examination at that time showed tenderness without spasm in the epigastrium, and tenderness in the left upper quadrant. The blood was normal. A Graham test was positive, although no stones were visualized. A cholecystectomy was performed, and palpation of the common duct revealed no stones. The gastric area was firm and adherent. The pathological diagnosis was chronic cholecystitis. Postoperatively she continued to have abdominal pain. A fluoroscopic examination of the stomach three weeks postoperatively showed a 2-cm. projection on the anterior wall near the lesser curvature and 5 cm. posterior to the pylorus, over which no peristaltic wave seemed to pass. The films made at the time did not show this finding. Re-examination three weeks later was also inconclusive. In some positions there appeared to be a large ulcer on the posterior wall at about the junction of the middle and lower thirds. The rugae of the entire stomach were coarse and irregular.

A second exploratory laparotomy was performed seven weeks after the first operation. The viscera above the omentum appeared to be densely plastered together, and a preliminary Witzel jejunostomy was performed. The patient was placed on a Sippy diet for five weeks, without improvement. She was finally discharged after six months of hospitalization but readmitted three days later because of recurrence of pain. The gastric contents two weeks after readmission consisted mostly of bile, with no free acid and 20 units of combined acid.

The patient remained in the hospital until two weeks before transfer to this hospital. During that period of hospitalization she had lost 65 pounds. She claimed that she had as much epigastric pain as before operation, but its character was different. It appeared primarily on deep inspiration and radiated bilaterally along both costal borders to the shoulder and arm on the left side. The pain apparently had no relation to meals or to bowel movements. The latter were scanty, light colored and never bloody or tarry. She vomited only rarely. For three days before entry she had been free of pain.

Physical examination showed a well-developed woman in no distress. The heart and lungs were normal. There were an old appendectomy scar in the right lower quadrant, a left rectus scar, and a small cruciate scar in the left flank, all of which were well healed. There was tenderness in the upper abdomen, most marked in the epigastrium, which felt diffusely more tense and resistant than the rest of the abdomen; no definite masses were palpable. Pelvic examination was essentially negative.

The blood pressure was 140 systolic, 95 diastolic. The temperature was 98° F., the pulse 80, and the respirations 20.

The white-cell count was 7300, with 75 per cent neutrophils. The red-cell count was 2,120,000, with 7 gm. of hemoglobin. The urine was negative except for the sediment, which contained innumerable red cells and 6 to 8 white cells per high-power field. A blood Hinton test was negative. The blood nonprotein nitrogen was 22.5 mg. per 100 cc., and the protein 7.7 gm.; the chloride was 97 milliequiv. per liter. Gastric analysis revealed no free acid in the fasting sample and 39 units after histamine. A gastrointestinal series showed a normal esophagus. Along the lesser curvature and posterior wall of the body of the stomach and extending down nearly to the angulus was a 7-cm. area of rigidity, which produced a filling defect (Fig. 1). The edges of the latter were smooth. There was no definite shelf, and the mucosal pattern, although slightly irregular,

*On leave of absence.

appeared preserved over the greater portion of this area. No crater could be demonstrated, although one small irregularity was noted that could have been the edge of a fold or a small crater. The remainder of the stomach and the duodenum were not remarkable. Gastroscopy on the second day revealed a normal antrum that appeared to be directed somewhat anteriorly. The body and lower portions of the fundus were well visualized, and the mucosa was everywhere intact. There were some adherent secretions on the posterior wall high in the body, and the mucosa in this region appeared fixed. There was no inflammation of the gastric mucosa.

The patient was placed on a six-meal bland diet and was given morphine for pain. For the first two days she did well. On the third day she had considerable retching and vomiting and abdominal pain. She developed severe anorexia and was unable to take food by mouth. She was given intravenous fluids. The following day the pain became severer, radiating to both shoulders, the precordial region and the neck. A Levine tube alleviated the vomiting, and the pain was controlled by morphine. She was then given daily intravenous fluids and several transfusions.

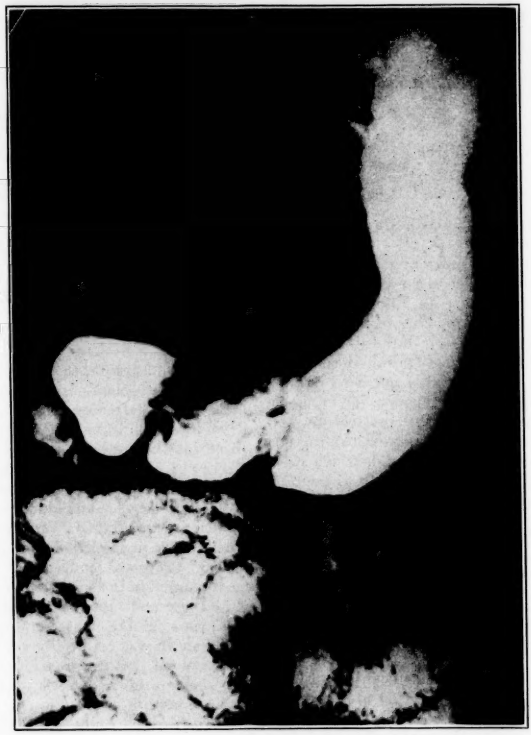

FIGURE 1. *Roentgenogram of the Stomach following a Barium Meal.*

On the tenth hospital day an exploratory laparotomy was performed.

DIFFERENTIAL DIAGNOSIS

DR. ROBERT R. LINTON: This thirty-nine-year-old patient was undoubtedly suffering from some form of gastric tumor. The original history quite obviously suggests gallstones, because she had pain in the epigastrium that radiated to the right scapula, and also associated with it she had one attack of jaundice.

So the surgeon who saw her originally had a right to suspect gall-bladder disease and cholelithiasis. She gave a story of having had copious vomiting. Copious vomiting in the presence of cholelithiasis alone is rather unusual and would have made me suspect that perhaps there was something in addition. I should also like to point out the fact that the Graham test was reported as positive, although no stones were visualized. It is our belief here — Dr. Robbins can correct me if I am wrong — that just because the gall bladder does not show by cholecystogram it does not mean disease of the gall bladder. It just means that the dye has not been absorbed in the biliary tract. I am never satisfied with the diagnosis of cholecystitis merely because of the absence of a gall-bladder shadow.

The operation bears out the fact that the gall bladder was not particularly abnormal. It was noted at that operation that she had some lesion in the region of the stomach, which apparently was not investigated too thoroughly. As a result of re-examination by x-ray she had a second exploratory laparotomy, and again the surgeon was unable to make up his mind what the trouble was in the stomach. They did a preliminary Witzel jejunostomy. I do not know why. We do a jejunostomy essentially for feeding purposes if we anticipate a major abdominal operation involving the stomach, so that we can get the patient into better condition. Possibly that is what was planned, but the patient was never in good condition for an operation.

Dr. Benjamin Castleman: A portion of the operative note from the other hospital is as follows:

> Under spinal anesthesia a left upper rectus incision was made and carried up to the sternum. The peritoneum, stomach, omentum and lesser curvature were all plastered down with edematous adhesive peritonitis. Below the omentum, however, there was no evidence of generalized peritonitis. The lesser peritoneum was opened by going through the gastrocolic ligament; this was found to be adhesive. There was no question of being able to do a gastrectomy. It was therefore decided that the best thing that could be done for the patient was to perform a Witzel jejunostomy for feeding purposes, thus by-passing the stomach.

Dr. Linton: When the patient was finally admitted here, the salient points about the laboratory examinations are that she had marked anemia and a hemoglobin of 7 gm. The urine contained many red cells, which we shall have to discount because we have no proof that it was a catheter specimen.

I should like to see the films of the gastrointestinal series that was done here.

Dr. Laurence L. Robbins: This is the area along the lesser curvature in which the defect was found. It is best seen on the spot films. You will notice that the mucosa appears to be intact on all the spot films. There is only one thing that suggests the possibility of a minute ulceration, but it has more the appearance of a fold of the mucosa on end rather than a crater. I do not see anything that suggests the shelflike margin of a carcinoma and certainly nothing that looks like a definite crater.

Dr. Linton: Examination of the stools bears that out, since they were negative for blood. Presumably there was no ulcer of the mucosa.

I am a little disturbed in making a diagnosis of tumor of the stomach in a patient who had had so much pain. I am unable to figure out why she had so much pain.

The first possibility is a gastric ulcer. That would explain the pain; but I do not see any evidence of it, and we have no other lead for that diagnosis. Apparently the pain was not affected by food, so I think that we can rule out ulcer on the history and also on the x-ray findings.

That leaves us with some kind of tumor of the stomach. The two that I am thinking of are a scirrhous type of carcinoma that had not ulcerated the gastric mucosa but was traveling in the submucosa and a leiomyoma or leiomyosarcoma. I believe that most leiomyosarcomas give a history of gastric hemorrhage, and this patient had no such history. The fact that she had lost a lot of weight and the fact that she had a marked secondary anemia make me think that she had a malignant lesion rather than a leiomyoma. For that reason I have to make the diagnosis of a scirrhous type of carcinoma of the stomach.

Dr. Francis D. Moore: Were films of the chest taken? The patient had so much pleural pain on both sides around the costal margins that it would be interesting to see what the chest showed.

Dr. Robbins: None were taken here. The chest was fluoroscoped during the gastrointestinal examination, and apparently nothing suspicious was observed.

Dr. Castleman: None were taken at the other hospital.

Clinical Diagnosis

Carcinoma of stomach.

Dr. Linton's Diagnosis

Carcinoma of stomach, scirrhous.

Anatomical Diagnoses

Subdiaphragmatic and peritoneal abscess, left, old.
Chronic adhesive peritonitis of upper abdomen, marked.

Pathological Discussion

Dr. Castleman: The operative note reads as follows:

> The scar of the paramedian incision was excised. There were several areas of fat necrosis in the wound. A hard fixed mass involved the stomach and pancreas. This was adherent to the liver as well as to the retroperitoneal structures. The whole process felt like extensive malignant disease. There was no apparent fat necrosis inside the abdomen itself. The origin of the tumor could not be determined.

The patient died six days following operation. At autopsy, on opening the abdominal cavity, we found, especially on the left side, large numbers of dense, tough, fibrous adhesions. They involved the whole upper abdominal cavity, particularly the left subdiaphragmatic region. When the adhesions on the left side between the inferior surface of the stomach and the abdominal wall were separated, a large abscess was broken into; this measured about 10 by 4 by 3 cm. and apparently was the mass that was felt at the previous operation. A few small abscesses were also found. Further dissection showed only extensive adhesions. The mucosa and wall of the stomach were perfectly normal.

What the fundamental disease was at the onset of symptoms, I do not know; but I should guess that most of the pain that followed the first operation was due to the abscess and the extensive adhesions that were being formed.

DR. MOORE: Was this a colon-bacillus abscess?

DR. CASTLEMAN: The abscess was not cultured. The wall was thick and made up of granulation tissue. It must have been there for a long time.

DR. LINTON: The abstract gives only one temperature reading, which was 98°F. Did she have fever at any time?

DR. CASTLEMAN: The chart was normal except for one rise to 101°F.

DR. LINTON: Do you think that this was a pancreatitis with abscess formation?

DR. CASTLEMAN: We sectioned the pancreas and could not find any evidence of disease. We considered that diagnosis at the time of autopsy but found no evidence of fat necrosis except in the abdominal incision, which one finds without pancreatitis.

DR. LINTON: She still might have had a perforated ulcer.

DR. CASTLEMAN: We looked carefully for evidence of an old ulcer but could not find even a suspicious area.

It might be interesting to read the interpretation of the radiologist, which is as follows:

> The findings are those of an area of rigidity along the lesser curvature of the body. It is possible that this represents intramural extramucosal involvement due to an inflammatory process as a result of a previously perforated ulcer. If one is to consider tumor, it would probably be one of the spindle-cell group. Suggest gastroscopy.

DR. ROBBINS: That was only because it had been reported to me that there had been an ulcer and I could not find it.

CASE 30312

PRESENTATION OF CASE

A forty-one-year-old Irish housewife was admitted to the hospital because of nausea and abdominal pain.

The patient had been in good health until about one year prior to admission, at which time she began to experience nausea in the morning unassociated with meals and wearing off by noon. There was no vomiting. She also noticed a tearing pain in the left upper quadrant of the abdomen on bending over. Eight months prior to entry she developed severe back pain, apparently in the region of the kidneys, coming on at night or during damp cold weather. She had had no hematuria or other urinary complaints. These attacks diminished as the weather became warmer. For about the same length of time she had noted a burning sensation over the left side of the face and extending downward to the left breast and flank. During this period she had increasing fatigue. The nausea became progressively worse and was aggravated by food and exertion. During the three months prior to admission she developed a burning sensation in the hypogastrium, associated with diminution in the burning over the face and chest, and a bearing-down sensation in the pelvis. On examination at another hospital she was told that her spleen was enlarged. She was referred to this hospital for study.

The family history except for renal disease in her mother, maternal aunt and maternal grandfather was noncontributory. The patient had worked in a rubber factory some years previously and had been exposed daily to benzol fumes over a period of eight years. For the three years prior to admission her menstrual periods had become irregular and scanty and she had been experiencing hot flashes, chilly feelings and bouts of sweating, all of which had diminished in intensity.

Physical examination on admission revealed a well-developed and well-nourished woman in no discomfort. A few prominent veins were present on the chest. Several fixed nodes were felt in the right axilla. The heart and lungs were normal. The spleen was palpable five fingerbreadths below the costal margin, and was firm and nontender. The liver was not felt.

The blood pressure was 120 systolic, 80 diastolic. The temperature was 99°F., the pulse 92, and the respirations 20.

Examination of the blood revealed a white-cell count of 3100, with 71 per cent neutrophils, 23 per cent lymphocytes, 5 per cent monocytes and 1 per cent eosinophils; there was some decrease in the platelets, and a definite shift of the neutrophils to the left, with numerous band forms. The red-cell count was 3,900,000, with 9 gm. of hemoglobin. The mean corpuscular volume was 97 millimicrons, and the mean corpuscular hemoglobin 22 millimicrograms. The urine was repeatedly negative. The sedimentation rate was 42 mm. per hour, and 0.65 mm. per minute (corrected). The serum protein was 6.2 gm. per 100 cc., with an albumin-globulin ratio of 1.2. The blood cholesterol was 128 mg., the calcium 12.4 mg., and the phosphorus 3.9 mg. per 100 cc. The prothrombin time was normal. A bromsulfalein test revealed only 10 per cent re-

tention of the dye. A van den Bergh test and the urine urobilinogen were normal. A red-cell fragility test was normal. An agglutination test for brucellosis was negative. Tuberculin tests in dilutions up to 1:100 were negative. A blood Hinton reaction was negative. A gastric analysis revealed 47 units of free acid thirty minutes after histamine.

An x-ray examination of the chest showed the right middle lobe to be slightly decreased in size, with areas of scarring and fibrosis; the remainder of the lung fields was clear. A plate of the abdomen revealed a markedly enlarged spleen, but the liver shadow was within normal limits. Roentgenographic examination revealed a normal esophagus. Examination of the femurs showed that the cortices of the lower portions of both bones were slightly thinner than normal, with a slight bulge about 10 cm. above their lower ends. The tibias, fibulas and humeruses were negative. A sternal bone-marrow biopsy revealed normal marrow.

On the twenty-sixth hospital day a splenectomy was performed.

Differential Diagnosis

Dr. Wyman Richardson: I am going to tackle this problem by asking the question, Why was splenectomy performed?

The reasons for doing splenectomy, aside from a surgical condition such as splenic abscess or ruptured spleen, are hemolytic anemia, especially the familial hemolytic anemia, in which it is very successful, thrombopenic purpura, in which it is successful in 85 per cent of the cases, and perhaps thrombosis of the splenic vein, in which, in my experience, recurrence of hemorrhage, the reason for doing the splenectomy, is frequent. What else might one do it for? One may also take out a big spleen, such as the spleen found in Gaucher's disease, or occasionally in malaria, simply because it is so big. Of these possibilities everything has been ruled out except thrombosis of the splenic vein, and we have no evidence that such a condition was present. I shall come back to that in a moment.

I shall now take up the case from the clinical side. At the risk of being repetitious, I shall say in the first place that I think a blood smear in this case would be a fundamental thing to have seen, because the blood picture was peculiar and there is no mention here regarding the red cells — whether there was evidence of regeneration and polychromatophilia. In reading the history one gets the impression that something quite generalized was going on that had little to do with the weight of a large spleen. There were nausea and difficulty with food, neither of which has been explained. There was a tearing pain on bending over. Was that the pain of a large spleen, or pain from a lesion in the back? It seems to me that this was the pain of a lesion of the spine, with involvement of nerve roots. There was a burning sensation over the left side of the face, which also involved the left side of the chest and flank — just where "the flank" is, I am never sure. How can one explain that? Was it on the basis of diffuse bone involvement, or was it a peripheral neuritis, or was it some sort of functional or emotional type of reaction? In addition, she had a bearing-down pain in the pelvis, which is hard to explain but may have been on the basis of some involvement of the nerve roots. The nausea is still unexplained.

Then we come to the question of exposure to benzol. I agree with Dr. Hunter that the safe amount of benzol to have in the air is zero. It is becoming more and more apparent that exposure to benzol can do anything to the blood picture. If there is exposure to large amounts of benzol, rather acute exposure, for weeks or months one finds the picture that we used to think was characteristic; that is, the process starts with granulocytopenia, goes on to thrombopenia and anemia and ends up with aplastic anemia or pancytopenia, an extremely serious clinical picture, which is sometimes relieved by transfusion. If there is exposure to minimal amounts of benzol over a long period of time one finds an entirely different picture: polycythemia, which is indistinguishable from true polycythemia, leukemia, which is indistinguishable from true leukemia, or finally a picture that has been referred to as "myeloid metaplasia" or extramedullary blood formation occurring in the spleen.

What about the picture we are presented with here? There was no granulocytopenia. In fact, the striking thing is that the leukopenia was a total leukopenia. This is not the picture that one sees with simply a congested spleen due to splenic-vein thrombosis or to portal cirrhosis, because when leukopenia results from a congested spleen it is almost always due to a reduction in the granulocytes. It is certainly not the picture that one sees with acute benzol poisoning. Furthermore, a bone-marrow biopsy was reported as negative. So, somewhat reluctantly, I am going to give up the idea that these findings were due to benzol poisoning.

Physical examination does not help us except for the fact that fixed nodes were felt in the right axilla. Apparently they did not make a great impression on the people taking care of this patient because nothing more is said about them.

Dr. Benjamin Castleman: I might read what one observer said: "In the axilla there appear to be enlarged lymph nodes. If they are nodes, they are fixed."

Dr. Richardson: That makes it a little different. I shall come back to the nodes in a minute.

Let me point out that the blood showed a macrocytic hypochromic anemia, which is striking and rather rare. When seen, it is usually in adults who have experienced blood loss for an unusually long period. There is no such story here, so that either the mean corpuscular volume was wrong or there were present in the blood smear large cells, probably

immature polychromatophilic cells, that would have raised the mean corpuscular volume. The sedimentation rate (0.65 mm.) was definitely elevated. The calcium (12.4 mg.) was high, and the phosphorus (3.9 mg.) was on the high side of normal. Why was the calcium high? My thought is that the calcium was high because there was a destructive bone lesion. The bone was being destroyed, and calcium was being excreted. The phosphatase level might have been interesting but not too helpful. There was plenty of free hydrochloric acid. The rest of the laboratory tests were negative. I have reluctantly ruled out benzol poisoning. Were there any varices?

DR. LAURENCE L. ROBBINS: No.

DR. RICHARDSON: I rule out thrombosis of the splenic vein on the basis of the clinical picture, the lack of esophageal varices and the lack of a history of bleeding. There is no evidence of cirrhosis of the liver unless the presence of a normal-sized liver and an enlarged spleen is suggestive.

Is there any evidence of bone tumor in these x-ray films?

DR. ROBBINS: Except for thinning of the cortex in the lower portions of the femurs, the bones are not abnormal.

DR. RICHARDSON: There was some talk about a bulge.

DR. ROBBINS: It is not particularly abnormal.

DR. RICHARDSON: What about the middle lobe of the right lung?

DR. ROBBINS: I should think that that was probably an old process, with perhaps some bronchiectasis.

DR. RICHARDSON: How about carcinoma?

DR. ROBBINS: I see no sign of a mass or anything that suggests complete collapse; it is only partial atelectasis.

DR. RICHARDSON: What I am coming down to as a diagnosis is a diffuse malignant process involving the bone marrow. In such cases one usually gets a leukocytosis instead of a leukopenia, but I have seen several cases in which there was this type of leukopenia. I would feel better about it if there were definite evidence of red-cell regeneration in the blood smear.

What kind of tumor was it? Could it have been a myeloma? The patient was young. The total protein was normal, although the globulin was perhaps slightly elevated — and the albumin was a little low. There is no other evidence of myeloma.

Lymphoma may give this picture; I have seen an identical case. That again brings up the question of the axillary lymph nodes. If this patient had enlarged nodes, I submit that an axillary biopsy should have been done before splenectomy.

DR. CASTLEMAN: Another observer said, "There is one large lymph node in the left axilla."

DR. RICHARDSON: If there were enlarged nodes in either axilla I should have taken them out before doing a splenectomy. We cannot, however, settle that question now.

What other tumors should be considered? Nephroma ought to be, but there is no real evidence for it. Carcinoma in the lung? There are no symptoms suggesting it. Carcinoma elsewhere, perhaps of the stomach or pancreas? It would seem that one should consider carcinoma of the stomach. I shall assume that that was ruled out, although it is not mentioned. Was a gastrointestinal series done?

DR. CASTLEMAN: There is no record of it.

DR. RICHARDSON: Of all the possibilities I have mentioned it seems to me that lymphoma is likeliest, but I should not be surprised if it were carcinoma. Again, I say that it is with reluctance that I give up the possibility that benzol played a part, but I do so definitely.

DR. J. H. MEANS: Did you mention any conditions for which one would not do splenectomy?

DR. RICHARDSON: The reasons that I mentioned for doing a splenectomy are hemolytic anemia, thrombopenic purpura, splenic-vein thrombosis and possibly a large spleen that is mechanically difficult to carry around, usually a Gaucher's spleen.

DR. MEANS: I simply wanted to be sure that I heard all your reasons because I came to grief standing on the same spot that you are standing on less than twenty-four hours ago in one of these exercises put on for the third-year class. Most of the students made the correct diagnosis, but I did not. One of the features in that case was a big spleen. I do not suppose that the same diagnosis holds in this case, but it is an interesting point to bring up in differential diagnosis of any large spleen.

DR. RICHARDSON: I also mentioned malaria.

DR. MEANS: It was not malaria.

DR. CASTLEMAN: Why not tell him what it was?

DR. MEANS: Sarcoid.

DR. RICHARDSON: I never thought of it.

DR. MEANS: Neither did I — yesterday.

DR. RICHARDSON: I do not believe that this patient had sarcoid.

DR. MEANS: Are those shadows around the lung roots normal?

DR. ROBBINS: From the film alone I think they are vascular shadows. Of course one would have to see them fluoroscopically. If they are vascular, they are within the limits of normal; if they are lymph nodes, they are prominent.

DR. RICHARDSON: I believe that this was some form of malignant tumor involving the bone marrow and that the likeliest guess is lymphoma.

DR. MEANS: Why did they take the spleen out?

DR. RICHARDSON: Why did they? I think that it was a great mistake to have taken it out.

DR. MEANS: Was the pathologist consulted on this? Was a frozen section done?

DR. CASTLEMAN: No.

DR. MEANS: I should like to take Dr. Richardson up on one point. I do not believe that lack of evidence of esophageal varices rules out splenic-vein occlusion. I had a famous case with numerous varices in the stomach that neither the radiologist nor the endoscopist could find, but the surgeon found them all right.

CLINICAL DIAGNOSIS

Splenomegaly.

DR. RICHARDSON'S DIAGNOSIS

Malignant lymphoma of spleen.

ANATOMICAL DIAGNOSIS

Sarcoid of spleen.

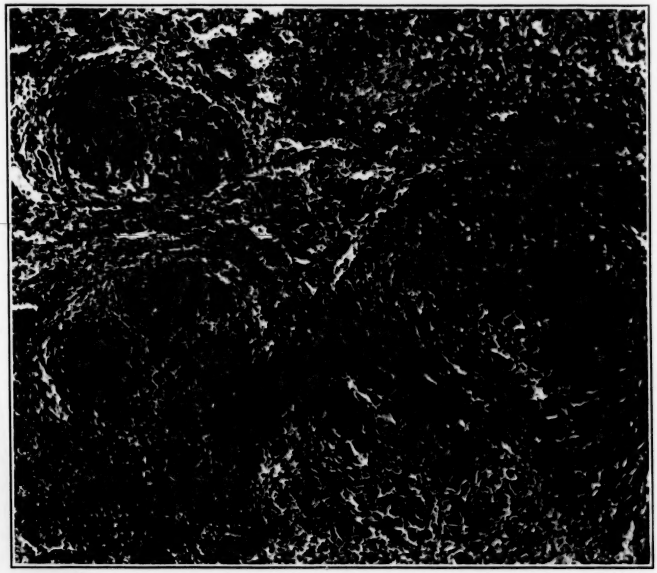

FIGURE 1. *Photomicrograph of Section of Spleen.*

DR. RICHARDSON: The patient we are discussing today did not bleed.

DR. MEANS: I meant merely that perhaps you are taking a risk in ruling out splenic-vein occlusion just because no varices were demonstrated.

DR. CASTLEMAN: Dr. William Dameshek was called in to see this patient. His note is as follows:

> This patient has a very large, hard spleen, associated with leukopenia and probably thrombopenia. There is no evidence of hemolytic anemia, cirrhosis of the liver or lymphatic leukemia. Nor does the blood or bone-marrow picture show any evidence of myelogenous leukemia or myeloid metaplasia. The hypochromic anemia may be coincidental and might respond to iron. Gaucher's disease appears to have been ruled out. This leaves us with either a chronic infectious splenomegaly (? type), a Boeck's sarcoid or a primary neoplasm of the spleen. I think that the spleen is bulky enough and causing sufficient symptoms to be removed.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: The spleen was about three times its normal size and was removed. Scattered throughout the parenchyma were round, ill-defined nodules 1.0 to 1.5 cm. in diameter, many of which produced a vague nodularity on the surface. Microscopically these proved to be lesions characteristic of sarcoid (Fig. 1).

The patient has been followed in the Out Patient Department for two years and is feeling well.

DR. RICHARDSON: What about the nodes?

DR. CASTLEMAN: They probably would show sarcoid. None were ever removed.

A chest plate taken a month or so ago shows no change.

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MEDICAL CERTIFICATION IN INDUSTRY

THE doctor has always regarded demands for written statements as burdensome. A Middlesex County fee table of one hundred years ago contains, among others, the following items:

For a visit in the village	$ .75
For a visit out of the village	1.00
For rising in the night and a visit, the night being considered as beginning at 10 o'clock and ending at sunrise	1.50–2.00
For a letter of advice	3.00

Were a corresponding value to be placed on the written statements the doctors of today are required to make most of them would prosper beyond their most optimistic dreams. In addition to reporting births, deaths, accidents and contagious and other diseases that come to their attention, the doctors are more and more called on to express opinions concerning all sorts of controversial situations. They must decide if the student with a "cold" is to be excused from the examination he is not too well prepared to face, if patients are entitled to extra food, fuel, or other rations, if various types of work are or could be prejudicial to the health of certain people and if other people are completely convalescent and able to return to work or merely wish to extend a period of incapacity into a vacation. The decisions themselves always have to do with individuals, and on this account alone the circumstances are never twice alike. The manner in which the profession is meeting these responsibilities has recently been causing some concern. It is a department of professional activity in which character and integrity count, as they so often do, more than skill and knowledge, and it is also a department in which the profession is closely watched by those who would encroach upon it from without.

A letter from an industrial physician appears in the correspondence column of this issue of the *Journal*. This doctor is looking on both sides of the problem of medical certificates, and is suggesting that those who have to do with such certificates do likewise. The War Participation Committee of the Massachusetts Medical Society has recently had its attention called to the matter, and has prepared a blank form to cover medical certification for those seeking new employment in essential industries; a statement by the committee and a copy of the blank also appear in this issue of the *Journal*. The prestige of the medical profession is therefore being widely invoked in areas that not only have a direct bearing on its own reputation and future but that are now concerned with the survival of democracy itself.

The doctor has traditionally been called on to be an advocate of his patient. It has usually been the individual who has employed and paid him, and he has instinctively defended the patient against impersonal groups whose interests have been more highly socialized. Although the dignity of the individual is one of the tenets of democracy, such groups as insurance companies, benevolent societies and uncoerced industrial interests and enterprises are equally essential elements of our national structure, and they, too, have claims on medical

science and opinion. Not all persons are above raising unfair and unjustified controversies between themselves and these impersonal groups, and many of them are clever enough to exploit the physician's instinctive desire to please his clients. Any question of illness or health that is raised becomes a crucial issue, and the doctor is called on to express his opinion, which if it is accepted becomes one of the determining factors in the outcome. If the doctor's judgment in these matters is favorably made and wise, the usefulness and reputation of the profession will be greatly enhanced. If, on the other hand, judgments are carelessly expressed, they may well become a means by which the profession can be undermined.

POLYPHARMACY AND ANESTHESIA

THE scientific advances in anesthesia in the last decade have been truly spectacular, and largely because of them, surgeons have been able to operate successfully in many regions of the body previously inaccessible. Furthermore, the safety of other operative procedures has been increased by these new methods, provided they are expertly applied.

A development parallel with these tremendous advances but one that bothers many surgeons is the use of multiple sedatives in connection with anesthesia when such sedatives can hardly be expected to add materially to the success of the operation. A case in point concerns a healthy seventeen-year-old boy with early, uncomplicated appendicitis. For reasons that seemed adequate, the surgeon elected to do the operation under spinal anesthesia, although local anesthesia, any one of the anesthetic gases and oxygen or plain ether would have been perfectly satisfactory. Preoperatively, the patient was given by injection 1/6 gr. of morphine, 1/200 gr. of scopolamine and 2 gr. of Sodium Luminal. (One wonders how the anesthetist knows that 1/200 gr. of scopolamine is going to contribute to the success of this procedure. Would not ¼ gr. or even ⅓ gr. of morphine achieve approximately the same effect? One also wonders, but not so seriously, whether the Sodium Luminal was necessary.) Spinal anesthesia, using a mixture of Pontocaine and procaine, was given easily and with immediate effect, and the operation proceeded. After fifteen minutes the anesthetist was busily engaged in giving artificial respiration by compression of the rubber bag on an anesthesia machine and administering oxygen and carbon dioxide through the machine, and a nurse was giving hypodermic injections of Coramine. The artificial respiration proceeded with a minimum disturbance, except mental, to the surgeon, who completed the operation. At that time the patient was still asleep and breathing fairly well, with shallow respirations at a rate of 12 to 15 per minute; he could be roused but immediately dropped off to sleep again. Half an hour later the spinal anesthesia was still complete to a level just above the nipples. Convalescence was without event. (One wonders why, if the anesthetist had confidence in his spinal anesthesia, he had to give in addition a practically fatal combination of sedatives.)

Just because the well-trained anesthetist can keep a patient going under conditions such as these hardly makes one believe that this amount of preoperative medication is necessary. Furthermore, in similar cases of collapse, resuscitation is not always successful.

Many surgeons regularly use nurse anesthetists in their hospital practice, much to the chagrin of the anesthesiologists. Nurses, of course, are not always perfect anesthetists, but when well trained they do excellent work and, in general, heavy premedication is avoided, since the surgeon gives the orders.

A great advance in anesthesia might well be achieved by conscious efforts to simplify sedation in the average easy case, which comprises over 90 per cent of surgery. If the anesthetist could limit himself to a moderate dose of a single sedative, together with a suitable dose of atropine or some substance having the pharmacologic effect of atropine, followed by a single gas, vapor or injection, the vocation of anesthesia might be raised from its present position, where there is perhaps a little too much art and not enough science. Until this is done, the surgeon will have to protect his patient by consulting with the anesthesiologist in detail concerning each step of medication given before, during and after operation.

OBITUARY

SIDNEY CHASE GRAVES

1901–1944

Lieutenant Commander Sidney Chase Graves, Medical Corps, U.S.N.R., died on May 14, 1944, in the Pacific area. No details concerning this tragedy are yet known except that he had been well but a few days before and that death did not occur in action. Until his departure earlier this year for an assignment overseas, he had been for eighteen months on duty at the United States Naval Hospital, Chelsea, Massachusetts.

SIDNEY CHASE GRAVES

Dr. Graves was born on July 21, 1901, at London, England, the first child of Alice Myrick (Chase) Graves and the late Dr. William Phillips Graves. Thereafter his life centered about Boston and Nantucket. He went to Noble and Greenough School, Boston, thence to St. Paul's School, Concord, New Hampshire, and prepared for college at Milton Academy, Milton, Massachusetts.

From Harvard College he entered the Harvard Medical School, receiving his degree in 1929. He acquired his clinical training first at the Massachusetts General Hospital and then at the Free Hospital for Women. He became a member of the staff of the latter in 1932, and was appointed assistant in gynecology at the Harvard Medical School in 1933. He carried on his private practice in Boston. He was a member of the American Medical Association, the Massachusetts Medical Society, the Boston Medical Library, the New England Obstetrical and Gynecological Society and the Obstetrical Society of Boston, a fellow of the American College of Surgeons and a diplomate of the American Board of Obstetrics and Gynecology.

In 1934 he married Alice Driver Brown of Philadelphia and is survived by her, their three children, his mother, a brother and a sister.

From his early school days his even disposition, cheerfulness and good comradeship made him the center of an admiring group of friends. As he progressed through school and college and reached maturity, these characteristics became more manifest, so that he was known and liked by an ever-increasing circle both within and without the medical profession. He excelled in many sports and developed considerable skill in sketching, his habitual "doodling" consisting of drawings of animals, faces and amusing caricatures.

In professional work Dr. Graves showed great earnestness and, one might say, a super-conscientiousness for the welfare of his patients, both private and free. Their devotion was his reward. The good results of his reliable surgery are constantly evident in the follow-up of his patients. For example, patients with tumors of the breast had been his special assignment for ten years at the Free Hospital for Women and the survival rates in cancer of the breast are now unusually high.

The happiness of his family life was obvious to all who entered his home and was affirmed in a tribute recently written by his wife. He was their "whole world." Those who knew him will remember him for his outstanding integrity, kindliness, humor, sociability and sportsmanship. He leaves his many associates with a feeling that he was robbed of many useful, happy years and that a true friend who can never be replaced was taken from them.

J. L. N. and G. V. S.

MASSACHUSETTS MEDICAL SOCIETY

WAR PARTICIPATION COMMITTEE

The accompanying certificate form (Fig. 1) has been adopted by the War Manpower Commission as the standard certificate for physicians to use when recommending change of employment for patients whose condition is or may be aggravated by continuance in their present jobs. The War Participation Committee has assisted in making this form as brief as possible in order to save physicians' time and to give the necessary information to the War Manpower Commission.

The War Manpower Commission is of the opinion that physicians have been too easily prevailed on to issue certificates for change of employment. It is urged that physicians issuing these certificates exercise considerable care and discretion to make sure continuance in the present job is harmful to the patient.

The War Participation Committee has agreed to review these doctors' certificates, much in the same way that Dr. Garland's committee reviews physicians' applications for extra rations from the various ration boards.

In summary, the War Participation Committee has agreed to assist the War Manpower Commission and to co-operate with it fully. The War Manpower Commission understands that our committee is interested in seeing that physicians are fairly and courteously treated by the district offices of the Commission. In other words, we stand ready to try to adjust any complaints physicians have against the War Manpower Commission or its branches, as well as to review the certificates sent to the Commission.

GUY L. RICHARDSON, *Chairman*

WORKER

Last Name | First | Middle

Address | Social Security Account No.

UNITED STATES EMPLOYMENT SERVICE of the WAR MANPOWER COMMISSION

MEDICAL CERTIFICATE

1. How long have you been treating the worker named above? _____

2. Diagnosis? _____

3. Would continuance of his present job be injurious to the worker? ☐ Yes ☐ No

(a) If "yes," why? _____

4. What sort of work will his condition permit? _____

Form _____ Date _____ Signature _____ (Attending Physician)

This certificate approved by the War Participation Committee of the Massachusetts Medical Society. It is subject to review by that committee.

FIGURE 1.

DEATH

SPARKS — Ernest E. Sparks, M.D., of Cochituate, died July 21. He was in his seventy-second year.

Dr. Sparks received his degree from the University of Vermont College of Medicine in 1902. He established the Cochituate civilian-defense medical center. He was a member of the American Medical Association.

His widow survives.

MISCELLANY

ALVARENGA PRIZE

The College of Physicians of Philadelphia awarded the Alvarenga Prize on July 14, 1944, to Dr. Gervase J. Connor, of the Department of Surgery, Yale University School of Medicine, for an outstanding study entitled "Anterior Cerebellar Function: An analytical study in functional localization in the cerebellum in dog and monkey." This will be published in the December, 1944, issue of the *Transactions and Studies of the College of Physicians.*

The Alvarenga Prize was established by the will of Pedro Francisco daCosta Alvarenga, of Lisbon, Portugal, an associate fellow of the College of Physicians, "to be awarded annually by the College of Physicians on each anniversary of the death of the testator, July 14, 1883, to the author of the best memorial upon any branch of medicine which may be deemed worthy of the prize." The College usually makes this award for outstanding published work and invites the recipient to deliver an Alvarenga Lecture before the College. The College occasionally, as in this instance, awards the prize for an exceptionally important manuscript submitted in competition.

CORRESPONDENCE

INDUSTRIAL MEDICAL CERTIFICATION

To the Editor: Not the least of the time-consuming chores given to the physician in modern society is that of certifying to this or that concerning his patients. Because of the physician's role among his fellowmen, he emerged early in our social development as the logical one to certify birth, to certify insanity and to certify the occurrence of contagious disease. With increased emphasis on public-health measures the physician has the task of examining food handlers and candidates for marriage for the purpose of certifying that these are ostensibly free of certain specific infections. The physician is deemed to be the logical citizen, in a democratic way of life, to perform these necessary functions.

With the rapid growth of the insurance principle during recent years, there has been an increase in the need for medical

certification. Health and accident policies depend to an appreciable extent, it would seem, on the knowledge and integrity of practicing physicians for actuarial soundness and for fairness in the disbursement of funds. Industrial organizations have used the insurance principle to provide various benefits for employes. Compensation statutes require medical judgments as bases to support legal judgments. It has been necessary to use the doctor's statement in an equitable enforcement of rationing while the nation is at war. As one contemplates the intricacies of modern society it is apparent that the end is not yet!

It is doubtful that the physician could or should avoid his function in verifying the existence and character of illness for legitimate ends. There is reason to believe that he should, in some instances, insist on more adequate compensation for the time spent in preparing factually accurate certificates. There is likewise reason to believe that some certificates could be shortened or modified without loss of pertinent information. To avoid them altogether does not seem possible.

While the nation is at war and struggling to use its manpower with maximum effectiveness, the need to limit absenteeism in industry has focused attention on the character of medical certificates. (Babey, A. M. Absenteeism. *J. A. M. A.* **122**:395, 1943.) The certificate should be a reliable guide in releasing prompt financial assistance for the ill worker. An instance has been reported (Current Comment. *J. A. M. A.* **122**:812, 1943) wherein a physician "issued a certificate to the effect that a worker was sick with gastroenteritis and was unable to work for a period of six days in the month of May, whereas investigation developed the fact that the person who submitted the certificate actually was working in another establishment and was attempting to obtain pay from two sources by requesting sick leave with pay from one of them."

From personal experience, a request for disability was recently honored on the basis of a physician's certificate stating that the worker was unable to perform even light work at the plant. The worker accepted his disability payments and spent several weeks pushing a wheelbarrow filled with rocks to landscape his residence property! It is true that the family physician risks an irate patient if the physician objects to signing whatever statement the patient requests. On the other hand, one may ask what degree of confidence and respect will the patient have in his doctor if the latter, unwittingly or not, falls in with such connivance?

There is a pitfall in another direction. In view of the marked emphasis given by industry and its advisers to matters of industrial hygiene, the worker with any complaint is likely to blame his working environment. He has had his attention called to this possibility, and it is altogether reasonable that wishful thinking will lead him to indict fumes, gases, vapors, dusts and the like. Actually many unskilled workers have no idea what, if any, exposure they have at their jobs. Such a worker in the role of patient may influence his family doctor to certify to a most astounding and untenable diagnosis. It would seem well to urge that physicians protect themselves against these pitfalls by using confirmatory sources of information. The plant and the plant physician are sources for checking facts. Other sources are the state and federal divisions of industrial hygiene. The exceptionally complete library on industrial health and hazards of the Division of Occupational Hygiene, Department of Labor and Industries, 23 Joy Street, Boston, is available to New England physicians.

Much medical certification is done well. These comments are made not in a spirit of censure but rather by way of directing attention to a potential danger to the good name of the profession. If we must sign certificates, may they invariably be as unbiased and factual as is humanly possible. Our jobs must be scientifically done. Industry depends on the physician for an answer to medical problems. Regardless of their difficulty there is no one who is better qualified to get the answers than is the physician. Therefore it is essential that the approach to these matters should be sound and of the highest ethical quality, so that traditions that have cast so much luster on medicine in the past shall continue to flourish in the future.

LEMUEL C. McGEE, M.D., *Medical Director*
Hercules Powder Company
Wilmington, Delaware

TEST FOR HYPERSENSITIVITY TO SULFONAMIDES

To the Editor: In the important and comprehensive editorial, "Cutaneous Sulfonamide Hypersensitization," in the May 18 issue of the *Journal* nothing is said about demonstrating hypersensitization. As the problem of sulfonamide hypersensitization is much broader than its cutaneous aspects, and since some of the general manifestations may be very serious, even fatal to the patient, it would be helpful to have a satisfactory test of a patient's sulfonamide sensitivity. For this the use of the drug as a patch or intradermal test has been unsatisfactory, since negative tests are frequent, in fact, almost the rule. There is a test, however, that is simple and that is claimed by its originator to be positive in the great majority of patients. This consists in injecting intradermally a small amount of serum from a patient who is taking a sulfonamide drug (Leftwich, W. B. An intradermal test for recognition of hypersensitivity to sulfonamide drugs. *Bull. Johns Hopkins Hosp.* **74**:26, 1944), with the production of a wheal if the patient tested is sensitive to the sulfonamide that the patient from whom the serum was taken had been receiving. The test is said by the author to be highly specific and reliable in the diagnosis of sulfonamide sensitivity. If this is confirmed, it will be extremely useful in the clinic in the differential diagnosis of conditions simulating sulfonamide-sensitivity effects, and particularly as a precautionary measure to prevent giving again a sulfonamide to a patient who previously had had it and had become sensitized to it.

HENRY A. CHRISTIAN
20 Chapel Street
Brookline 46, Massachusetts

BOOK REVIEWS

Manual of Fractures: Treatment of external skeletal fixation. By C. M. Shaar, M.D.; and Frank P. Kreuz, Jr., M.D. 8°, cloth, 300 pp., with 148 illustrations. Philadelphia: W. B. Saunders Company, 1943. $3.00.

This manual is the outgrowth of the authors' experience in the use of the Stader splint for 157 patients with various types of fractures. They give a concise and lucid description of the types of fractures best suited to this apparatus, the technic of its application, the postoperative care and the hazards incident to its use. It has been found to be particularly useful in cases in which there are extensive burns or lacerations in the area of the fracture. This method of external skeletal fixation permits complete freedom of the joints, which in turn lessens circulatory disturbances and muscular atrophy. Early use with rigid fixation also hastens bony union. In battle casualties its use facilitates early and easy evacuation from danger zones. The authors have observed no infection from the pins that hold the fragments. They advise gentle impaction with the turnbuckle bar after the fracture is reduced. The treatment of different types of fractures, for example, that of the jaw, is taken up in detail. The apparatus, however, has been found most useful in fractures of the long bones. It has been used successfully in infected compound fractures and for fixation after bone grafts and arthrodesis. Chapters are appended on anesthesia in the treatment of fractures and on the roentgenologic aspects of fracture healing. The book is well illustrated with clear photographs.

Although this type of treatment of fractures will probably never become popular outside large hospitals, familiarity with such methods is desirable for all orthopedic and industrial surgeons. It is recommended as an interesting and useful book to those who are concerned with the treatment of fractures.

Geriatric Medicine: Diagnosis and management of disease in the aging and in the aged. Edited by Edward J. Stieglitz, M.S., M.D. 8°, cloth. 887 pp., with 187 illustrations. Philadelphia and London: W. B. Saunders Company, 1943. $10.00.

Geriatrics is becoming more and more a specialty of medicine since the number of older persons in this country has

been rapidly rising with the increased expectancy of life that has resulted from advances in both curative and preventive medicine. It is stated in the foreword that it will not be long before one third of the population of the United States will be over fifty years of age, and it has been estimated that within forty years there will be twice as many people living at more than sixty-five years than there are now. It is within the realm of possibility that the activities of pediatricians will become less whereas those of geriatricians will become more, although this is not an immediate probability as wartime marriages are raising the birth rates in many communities. With the constant reduction in infectious processes, the number of persons living into the later decades will be greatly increased and therefore there will be relatively larger numbers of cases of circulatory diseases with their varied manifestations.

The editor discusses geriatrics ably and at some length, and makes clear the fact that there is a wide difference between the aged and the aging. This is a point that some physicians and many of the laity overlook. An aged individual is one who has lived a considerable number of years and has therefore become, in common parlance, an old person. But an old person may have fewer signs of aging than one many years younger. Aging begins in the early years, and consequently the physician should have the objective and subjective signs of aging constantly in mind. That is one of the strongest pleas for the routine, regularly spaced, physical examination. To treat only those ill with a definite disease is, after all, rather poor medicine.

The reviewer has read several books on geriatric medicine, and although all are more or less treatises on general medicine, the present volume adheres more closely to the subject of aging than do any of the others. The editor has selected fifty-four authorities who have written with clarity on many important topics concerning the aging and the aged. Aside from excellent discussions of circulatory, lung, bone, joint, urogenital, blood and metabolic diseases, there is much sound advice to assist the general practitioner in readjusting the aging to the changing conditions of their lives.

A Handbook of Medical Library Practice, including Annotated Bibliographical Guides to the Literature and History of the Medical and Allied Sciences. Based on a preliminary manuscript by M. Irene Jones and compiled by a committee of the Medical Library Association. Edited by Janet Doe. 8°, cloth, 610 pp. Chicago: American Library Association, 1943. $5.00.

This pioneer work is a distinct contribution to the literature of medical librarianship. In the preface the editor states that the objective of the book is to discuss "library procedures, methods and subjects commonly used and found in medical libraries but different from those found in general libraries." These objectives are met, and the book is a manual of procedure, as well as a reservoir of useful data, for medical librarians. Its faults, mostly of omission and inadequate treatment of subjects, do not detract from the value of the book to the physician. They are technical in character and only affect the professional library worker.

The work is divided into eight chapters, each written by a member of the Medical Library Association. The first chapter is a general one and has to do with the medical library, its development, its distribution and its administration. Appended to this chapter are items of importance: the first a list of all medical libraries, situated anywhere in the world, having 100,000 or more volumes, and the second a description of medical libraries located outside North America. The other chapters are more technical and discuss such subjects as periodical and book selection, cataloguing, subject headings, classification, pamphlets, pictures and microfilms. The final chapters in the manual deal with rare books and the history of medicine, as well as reference work, including an annotated list of reference books. The essay on rare books and the history of medicine might well have been published separately, since it is more comprehensive than what the librarian of the average medical library needs. A shorter presentation of the subject, more dogmatic in character and featuring certain primary objectives, would have served the purpose better than the extended treatment indulged in by the author. The same can be said about the chapter on reference work. Much of the material in this section, however, is of particular interest to medical collectors, historians and bibliographers.

The outstanding weakness of the book is the failure of the various authors to discuss the fundamental values of the various procedures and methods treated. Nowhere can be found any reference to the relative importance of the various topics. The student of medical librarianship and the inexperienced library worker in the field of medicine is left adrift, since little attempt is made to suggest the best procedures to be followed in her daily work. Some subjects are presented, moreover, without indication why they are included.

The editing is well done, and the level of writing is uniform for a collected work, although there are places where the English could have been improved.

The Principles and Practice of Industrial Medicine. Edited by Fred J. Wampler, M.D. 8°, cloth, 579 pp., with 42 illustrations and 36 tables. Baltimore: The Williams and Wilkins Company, 1943. $6.00.

This is a selected group of thirty-three dissertations, each of which deals with a specialized problem in industrial medicine. It is not an elementary textbook.

Occasionally one of the contributors takes the bit in his teeth and runs away with his subject matter in a way that may well overwhelm the casual seeker after simple advice. It is possible to spread confusion rather than enlightenment by means of studies that are too erudite and elaborate. As a minor illustration, Dr. Foulger, of the Haskell Laboratory of Industrial Toxicology, finds it necessary to interpret blood-pressure readings on chemical workers with the aid of the formula,

$$\ln S = -0.00339\ (x^2+y^2),$$

where ln S is the logarithm of S (the score) to the base of 10, x the deviation of the diastolic blood pressure from an established mean of 79 mm., and y that of the pulse pressure from an established mean of 44 mm. This formula may be valid, and the conclusions reached in his study are interesting and probably significant, but a simpler presentation and interpretation would be more generally valuable in a textbook.

Topics covered include a variety of intoxications, the effects of temperature and humidity, the eyes, the back, compensation, women in industry, fatigue, the physical examination, the lay-out of medical departments and so forth. It is sadly ironic that the author of the excellent chapter on the cause and prevention of accidents should have been accidentally killed before his contribution appeared in print.

This book is recommended for reference reading.

Plastic Surgery of the Breast and Abdominal Wall. By Max Thorek, M.D., LL.D. With an introduction by Rudolf Nissen, M.D., and a foreword by J. Eastman Sheehan, M.D. 4°, cloth, 446 pp., with 458 illustrations. Springfield, Illinois: Charles C Thomas, 1942. $6.00.

The main subject of this book is the treatment of the hypertrophied breast. The author has made a sincere effort to describe the various operative procedures advocated by the many surgeons who have taken a special interest in this field of surgery. He analyzes quite extensively the different operative procedures that seem best suited to each particular problem. There is included a short chapter on reconstructive surgery of the abdominal wall, and also chapters on the anatomy, physiology and pathology of the hypertrophied breast.

The book is profusely illustrated with good diagrams and photographic illustrations, and it is one of the most complete monographs on the subject that has come to the reviewer's attention.

Convulsive Seizures: How to deal with them. By Tracy J. Putnam, M.D. 12°, cloth, 168 pp., with 12 illustrations. Philadelphia: J. B. Lippincott Company, 1943. $2.00.

This is a brief manual covering various types of convulsive diseases, particularly written for patients and their families and friends. There is a good discussion on seizures and their causes, with additional data on diagnosis and the principles involved in treatment. Excellent advice is given

regarding various drugs, and this part of the book is distinctly valuable to physicians. The final chapter deals with legal problems connected with epilepsy, and there is an up-to-date reference to the current literature in an adequate bibliography.

This is a book that may be put in the hands of the more thoughtful patient and will be of value to a considerable proportion of practitioners who occasionally have to make decisions in regard to epilepsy.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Backache and Sciatic Neuritis: Back injuries—deformities—diseases—disabilities. By Philip Lewin, M.D., associate professor of bone and joint surgery, Northwestern University Medical School, attending orthopedic surgeon, Michael Reese Hospital and Cook County Hospital, professor of orthopedic surgery, Cook County Graduate School of Medicine, Chicago, and lieutenant colonel, Medical Corps, United States Army. With line drawings by Harold Laufman, M.D., instructor in surgery, Northwestern University Medical School, Chicago, and captain, Medical Corps, United States Army. 8°, cloth, 745 pp., with 235 illustrations. Philadelphia: Lea and Febiger, 1943. $10.00.

This new book is a comprehensive treatise on diseases and injuries of the back. Emphasis is placed on diagnosis and treatment; the medicolegal, industrial and military aspects are also discussed.

Pathology and Therapy of Rheumatic Fever. By Leopold Lichtwitz, M.D. With a foreword by William J. Maloney, M.D., LL.D., F.R.S. (Edin.), consulting neurologist to the City Hospital, New York City. Edited by Major William Chester, M.C. 8°, cloth, 211 pp., with 69 illustrations and 11 tables. New York: Grune and Stratton, 1944. $4.75.

This monograph discusses rheumatic fever in all its acute and chronic aspects.

Internal Medicine in General Practice. By Robert P. McCombs, M.D., lieutenant, Medical Corps, United States Naval Reserve. 8°, cloth, 694 pp., with 114 illustrations. Philadelphia: W. B. Saunders Company, 1943. $7.00.

This new manual on internal medicine is written especially for practitioners and students, and the field of diagnosis and treatment is especially emphasized. All the newer methods and mechanical aids in diagnosis have been included.

The Youngest of the Family: His care and Training. By Joseph Garland, M.D., physician to the Children's Medical Department, Massachusetts General Hospital, consulting pediatrician, Massachusetts Eye and Ear Infirmary, and instructor in pediatrics, Harvard Medical School. Revised edition. 12°, cloth, 182 pp., with 11 illustrations. Cambridge, Massachusetts: Harvard University Press, 1943. $2.00.

This is a revision of a manual first published in 1932. It has been written to meet the exigencies of wartime. Because of the difficulty of obtaining many of the luxuries that once seemed indispensable in the care of the baby, instructions have been reduced to the simplest terms. Dr. Garland is a well-known pediatrician, and this revised edition should prove to be even more useful and valuable to the mother than the former edition.

Medical Genetics and Eugenics. Volume 2. By R. Ruggles Gates, Ph.D.,D.Sc.,Ll.D.,F.R.S., Marine Biological Laboratory, Woods Hole, Massachusetts; Laurence H. Snyder, B.S. M.S.,Sc.D. professor of medical genetics, Ohio State University; and Earnest A. Hooton, Ph.D.,Sc.D., Department of Anthropology, Harvard University. 8°, cloth, 60 pp., with 13 illustrations. Philadelphia: Woman's Medical College of Pennsylvania, 1943. $1.00.

The present volume includes lectures given before a public audience at the College of Physicians, Philadelphia. They have to do with the subject of heredity.

Fractures and Dislocations for Practitioners. By Edwin O. Geckeler, M.D. Third edition. 8°, cloth, 361 pp., with 320 illustrations. Baltimore: The Williams and Wilkins Company, 1943. $4.50.

This book was first published in 1937. The third edition has been revised to include sections on emergency treatment and fracture wounds, particularly vital at the present time. New procedures have been simplified and considerable attention has been given to chemotherapy in the treatment of traumatic wounds. Débridement in the treatment of compound fractures is described so that the physician can perform the operation without delay if an experienced surgeon is not available. The manual is primarily written for the general practitioner who may be required to treat traumatic injuries in the absence of a competent surgeon.

Prelude to Sanity. By S. Greiner. 8°, cloth, 164 pp. Ft. Lauderdale, Florida: Master Publications, 1943. $3.00.

It is the author's contention that, contrary to current psychiatric belief, schizophrenia does not constitute a clinical but a generic phenomenon.

Ship's Doctor. By Rufus W. Hooker, M.D. 8°, cloth, 279 pp. New York: Whittlesey House, 1943. $2.50.

This is the autobiography of a physician who for fourteen years followed the practice of medicine at sea, sailing over a million miles in that time. The book is written in a narrative style and relates many experiences of Dr. Hooker in varying situations, ranging from the urgent call of an SOS to convoying the wounded from Pearl Harbor and the Philippines.

The 1943 Year Book of Industrial and Orthopedic Surgery. Edited by Charles F. Painter, M.D., orthopedic surgeon to the Massachusetts Women's Hospital and Beth Israel Hospital, Boston. 12°, cloth, 440 pp., with 306 illustrations. Chicago: The Year Book Publishers, 1943. $3.00.

This volume provides a summary of progress for the year in the fields of orthopedic surgery and of industrial medicine and surgery. The material is well selected and printed with excellent type on a good grade of paper.

Nascent Endocrine Therapy. By John F. Ritter, M.D. 8°, cloth, 317 pp. Caldwell, Idaho: The Caxton Printers, Limited, 1940. $5.00.

In this book the author describes his treatment of essential hypertension.

Medical Advice from a Backblock Hospital. By G. M. Smith, M.B., Ch. B., F.R.C.S.E. With articles and notes by N. Bedggood, Vernon Brown, F. Cook, L. M. Copley, N. Giles, E. Harwood, A. Kernihan, W. H. Kirkpatrick, E. Tait, W. Maxwell and Barbara Orr. Second edition. 8°, paper, 96 pp. Wellington, New Zealand: Progressive Publishing Society, 1943. 1 shilling.

This work is a description and an explanation of the Hokianga Co-operative Clinic Medical Service, located in New Zealand. The first edition was published in March, 1942, and the second edition affords the author an opportunity to revise and add to his work and also to answer his critics. The principal part of the pamphlet is a manual of domestic medicine, to which are added replies to critics on social medicine among the Maoris.

Child Welfare Handbook: A guide to health and social services. Edited by Beatrice S. Stone. 8°, paper, 58 pp. Boston: Publications Committee of the Massachusetts Child Council, Incorporated, 1943. 25 cents.

This pamphlet is fundamentally a directory of all organizations in Massachusetts having to do with child welfare. It is an essential reference source of information concerning the subject.

(*Notices on page xiii*)